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WADC TECHNICAL REPORT 55-165

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WRIGHT AIR DEVELOPMENT CENTER

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Date Due

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FOREWORD

This report covers work carried out by the Department of Medicine, Duke University School of Medicine, Durham, North Carolina, under contract AF 33(616)441, with the Aero Medical Laboratory, Wright Air Development Center. This contract was in support of Project 7160, "Research in High Altitude Physiology," Edwin G. Vail, Captain, USAF, contract monitor.

ABSTRACT

This report covers three topics relating to the Seeler resuscitator.

Equations have been developed by means of which it is possible to describe a complete respiratory cycle of the resuscitator and to examine the results of change in the major variables of the subject resuscitator system. Particular attention has been devoted to the problem of providing adequate ventilation to a person with a high respiratory resistance.

The successful use of the Seeler resuscitator in cases of carbon dioxide narcosis is described. These patients have a high resistance and a low pulmonary compliance. Since they simulate in this respect persons with "nerve gas" poisoning, they are valuable subjects for testing the effectiveness of a resuscitator.

Some observations are made on the effects on arterial blood pressure and cerebral venous blood oxygen of inducing hypocapnia by over ventilation during resuscitation.

PUBLICATION REVIEW

This report has been reviewed and is approved.

FOR THE COMMANDER:

JACK BOLLERUD
Colonel, USAF (MC)
Chief, Aero Medical Laboratory
Directorate of Research

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SECTION I

A THEORETICAL STUDY OF THE OPERATION OF THE SEELER RESUSCITATOR

INTRODUCTION

Evaluating a resuscitator for use with different kinds of subjects requires an understanding of the major variables involved and the effect of changes in each of these on the subject resuscitator system. For the Seeler resuscitator the variables are line pressure, inflow resistance of the resuscitator, and the mask pressure range. For the subject the principal variables are the compliance and the resistance. As an aid to our evaluation of the resuscitator, an analysis of the subject resuscitator system has been made in terms of these variables. The approach applies in general to resuscitators of this type. The numerical results obtained depend upon the characteristics of the particular valve used in the study. This was a special low resistance, short expiration time valve.

ASSUMPTIONS

For the purposes of this study a number of simplifying assumptions have been made. The major assumptions are similar to those made in the analysis of Radford (1), to which the present work is much indebted. Some further assumptions are based on the particular characteristics of the Seeler resuscitator used in the study. Of necessity, the analysis is narrowly confined by these assumptions, and it can predict the real-life behavior of the resuscitator only in general terms. The principal assumptions are these:

1. The line pressure is constant.

2. In terms of resistance and compliance the subject functions as a single system. That is, there is only one compliance and one resistance within the system. From recent studies on intrapulmonary gas mixing in man (2) it is clear that this is not true for subjects with pulmonary disorders and is even inexact for normal persons. The compliance of the upper airway and the mask is taken to be zero.

3. The compliance and resistance are constant during the cycle. Actually, they both change with change in lung volume (1, 3). The resistance is especially variable and increases as the lung volume decreases, particularly when the resistance is already abnormally high.

4. Pressure-flow relationships for both subject and valve are such that $P = k\dot{V}^2$, where P is the pressure responsible for a flow of gas, $\dot{V}$ is the rate of flow, and k is a constant. This is a familiar assumption for gas flow through a respirator (1, 4), and

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its justification for the Seeler Valve will be presented below. For normal subjects at the usual resting flow rates it appears to be a satisfactory assumption that pressure and flow have a linear relationship ($P = k\dot{V}$) (1). For subjects with a pathologically high resistance, who are of primary interest in the present study, our preliminary results suggest that the expression, $P = k\dot{V}^2$, describes the findings better than the linear relationship. Actually, if the resistance is normally low, there is very little difference between the results obtained with the two expressions under the circumstances of this study. Some comparative data bearing on this point are presented later.

The data relating to inspiratory pressure-flow relationships of the resuscitator are presented in Figure 1. The expression $P = k_i\dot{V}^2$ evidently fits the data reasonably well. The mean values for k_i (inspiratory resistance constant) are:

with the resistance fully open (Fast), $k_i = 85$ cm. $H_2O/(\text{liter/sec.})^2$

and with the resistance fully closed (Slow), $k_i = 645$ cm. $H_2O/(\text{liter/sec.})^2$.

Expiratory pressure-flow relationships of the resuscitator are complicated because expiration is assisted by a venturi. In Radford's general analysis of the resuscitator subject system (1) it was necessary to deal with this problem by making the assumption that expiratory time would be equal to inspiratory time. In many situations this is far from the case. The problem is simpler for the present analysis because attention is confined to one resuscitator type. Fortunately it was possible to find a usefully simple expression for expiratory pressure-flow relationships at the two extremes of inspiratory resistance ("Fast" and "Slow") and for different line pressures. The expiratory pressure-flow pattern depends upon inspiratory valve resistance and line pressure because these factors control flow rate through the venturi. Figure 2 presents the expiratory pressure-flow values obtained experimentally at the two different inspiratory resistances and at several line pressures. When there is no flow through the venturi (line pressure = 0), the data fit the expression $P = ke\ \dot{V}^2$, where $ke = 21$ cm. $H_2O/(\text{liter/sec.})^2$. When the venturi is operating, the pressure-flow curves assume a more horizontal position, indicating that the effective expiratory resistance is increased. Over a portion of their length the pressure-flow curves for each of the two inspiratory resistance settings (Fast and Slow) tend to run roughly parallel to each other. This approximation is obviously grossly incorrect for the two lower line pressures on the "Fast" inspiratory resistance chart at mask pressures above 10 cm. H_2O, and, in general, it applies less well at high than at low mask pressures. This rough tendency of the pressure-flow curves for different line pressures to run parallel at a particular setting of the inspiratory respirator resistance suggests that the effective expiratory resistance at moderate mask pressures may depend upon the inspiratory resistance setting much more than on the line pressure. It is also evident from the graphs that the negative pressure which assists respiration varies, as expected, with the line pressure at a particular inspiratory resistance setting.

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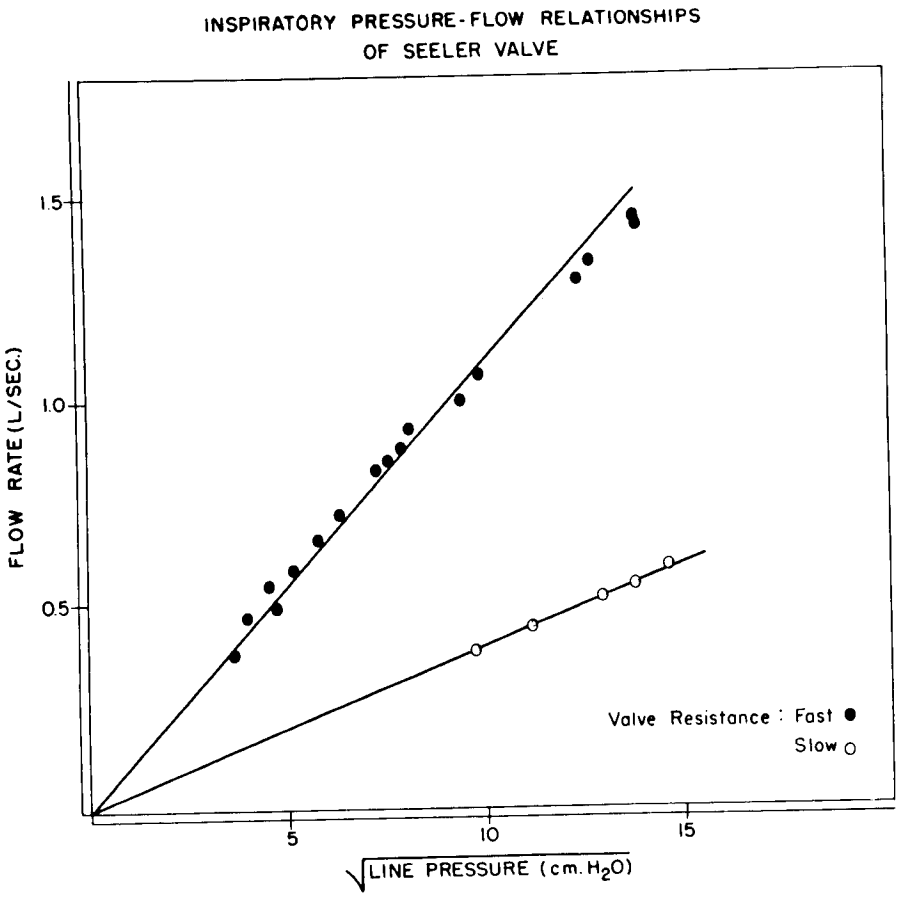

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Figure 1. It is apparent that the expression, Line pressure = k(Flow rate)^2, fits the data well.

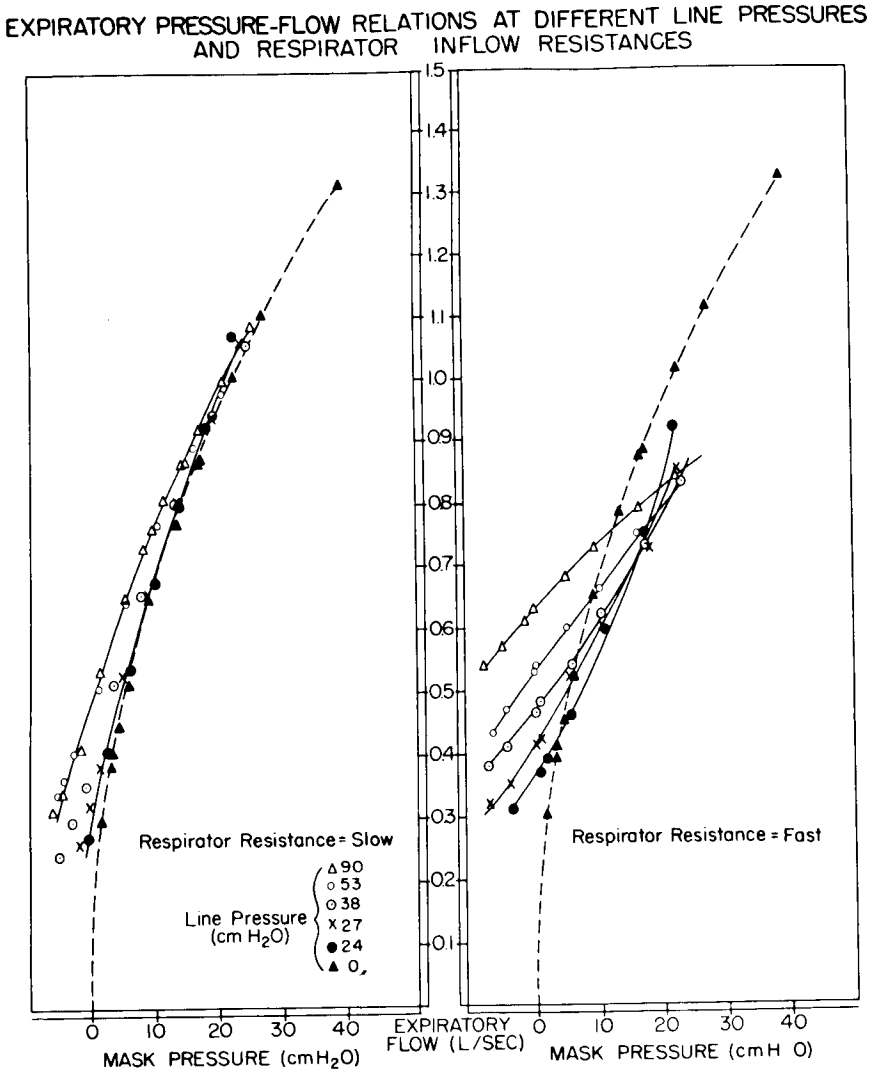

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Figure 2. The dotted lines show the pressure-flow relation for expiration through the resuscitator when the line pressure is zero, i.e. the venturi is not operative. The solid lines show the relation of expiratory pressure and flow when the venturi is operating at various line pressures. The left hand chart shows the situation at maximum inflow resistance. Solid lines are drawn only for the lowest and highest line pressures. On the right is presented the situation at minimum inflow resistance. It is apparent that at minimum resistance (right hand chart) and high flow rates the venturi will hinder expiration. At lower flow rates it will assist.

These considerations indicate that the expiratory pressure-flow curves at a given inspiratory resistance setting may be approximated by the expression

$$P - P_V = ke\ \dot{V}^2,$$

Where P is the mask pressure, P_V is the effective pressure produced by the venturi at the exhaust side of the resuscitator, ke is a constant, and $\dot{V}$ is the expiratory flow rate. For a given inspiratory resistance and line pressure the equation can be solved for P_V and ke by substituting values of P and $\dot{V}$ experimentally determined at two points. For the group of curves produced at the "Fast" setting, the points selected were, on the one hand, at zero mask pressure and, on the other, at positive mask pressures ranging from 20.5 cm. H_2O for 90 cm. line pressure to 11 cm. H_2O at 27 cm. line pressure. For the curves at the "Slow" setting the choice was substantially similar. The values obtained in this way for P_V and ke at the different line pressures are presented in Tables 1 and 2.

TABLE 1.

EXPIRATORY RESISTANCE, ke, AND VENTURI PRESSURE, P_V, AT DIFFERENT LINE PRESSURES: "FAST" INSPIRATORY RESISTANCE

Line Pressure (cm. H_2O)	ke (cm.H_2O/(L/sec)^2)	Pv (cm.H_2O)	0.33 Line Pressure (cm. H_2O)
0	21	0	0
27	60	-10	9
38	64	-14	13
53	62	-18	18
90	70	-28	30

Mean ke, venturi operating: 64 cm. H_2O/(L/sec)^2

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Within the limitations discussed above the expiratory resistance constant, ke, is usefully stable over a wide range of line pressures at a given inspiratory resistance setting. In addition, the effective venturi pressure at a given resistance setting can be described for present purposes as a constant fraction of the line pressure. For the "Fast" setting the effective venturi pressure is estimated as -0.33 times the line pressure, and at the "Slow" setting as -0.1 times the line pressure. The deviation from these approximations which occurs at the "Fast" setting when line pressure is low and mask pressure is relatively high results in a shorter expiration time and a greater minute ventilation than predicted by the equation.

TABLE 2.

EXPIRATORY RESISTANCE, ke, AND VENTURI PRESSURE, P_V, AT DIFFERENT LINE PRESSURES: "SLOW" INSPIRATORY RESISTANCE

Line Pressure (cm. H_2O)	ke (cm. H_2O/(L/sec)^2)	P_V (cm. H_2O)	0.1 Line Pressure (cm. H_2O)
0	21	0	0
24	23	-1.8	2
27	28	-3.5	3
38	29	-4.6	4
53	27	-6.0	5
90	27	-6.8	9

Mean ke, Venturi operating: 27 cm. H_2O/(L/sec)^2

The use of these approximations for the expiratory resistance constant and the effective venturi pressure allows calculation of the expiratory time under a variety of conditions.

Examination of Figure 2 shows that operation of the venturi at a "Fast" setting actually impedes expiration until the mask pressure falls to relatively low levels. At high mask pressures the fastest expiratory flows occur at zero line pressure, i.e. with the venturi inoperative. On the "Slow" setting the venturi always assists expiration.

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DERIVATION OF EXPRESSIONS DESCRIBING BEHAVIOR OF PATIENT-RESPIRATOR SYSTEM.

The purpose of the analysis is to describe the functioning of the patient-respirator system, within the limits of the assumptions, and given the values of the independent variables. Descriptions will be elaborated for two cases: that in which pressure-flow relationships of the patient obey the expression, $P = k \dot{V}^2$, as postulated in the present study, and that in which they obey the expression $P = k \dot{V}$, as postulated by Radford (1). The situation for $P = k \dot{V}^2$ is developed immediately below.

Symbols and conditions

Let Q = volume of gas in liters (ambient temperature and pressure, (usually 745-755 mm. Hg) saturated) added to the lung during the inspiratory portion of the cycle. No allowance is made for change in temperature or water vapor content as the gas is passed from the source through the respirator, into the lung.

Let A = Elastance (1/compliance) of lung in cm. H_2O/liter.

Pp = The intrapulmonary gas pressure (gage pressure, in cm. H_2O) owing to the elastance of the lung and chest wall. When Q=0, Pp=0. At other values of Q, Pp=QA.

Po = "Line pressure" (cm. H_2O), or pressure at which gas is supplied from the source to the resuscitator.

Pm = Mask pressure in cm. H_2O.

Pmi and Pmx are the mask pressures at which inspiration and expiration, respectively, are interrupted by the cycling of the Valve.

Pii and Pix are the initial mask pressures (cm. H_2O) at the start of inspiration and expiration respectively. Mask pressure is a discontinuous function at the point where the Valve cycles. To begin inspiration, the mask pressure passes discontinuously to Pii from the value it had when the Valve cycled to terminate expiration. At the end of inspiration, the mask pressure passes discontinuously to Pix for the beginning of expiration.

ki and ke are the inspiratory and expiratory Valve resistances in cm. $H_2O/(liter/sec)^2$.

k = The patient resistance in cm. $H_2O/(liter/sec)^2$, as postulated in the present study.

ti = Duration of inspiration in seconds.

tx = Duration of expiration in seconds.

F = Respiratory rate in cycles/minute.

$\dot{V}$ = Ventilation rate in liters/minute (ATPS). For calculating "effective" ventilation rate, a dead space of 200 ml. is postulated. The effective ventilation rate = $\dot{V}$-0.200 F, in liters/minute.

aPo = Effective venturi pressure. At F, a is -0.33 and at S, -0.1.

The basic equations

The relations between pressure, volume, and flow rate for patient and respirator which have been described above may be expressed as follows:

$$Pp = QA \tag{1}$$

For inflow through the respirator:

$$\left(\frac{dQ}{dt}\right)^2 ki = Po - Pm \tag{2}$$

For inflow into the patient:

$$\left(\frac{dQ}{dt}\right)^2 k = Pm - QA \tag{3}$$

For outflow from the patient:

$$\left(\frac{dQ}{dt}\right)^2 k = QA - Pm \tag{4}$$

For outflow through the respirator:

$$\left(\frac{dQ}{dt}\right)^2 ke = Pm - aPo \tag{5}$$

INSPIRATION

Q and Pm

From (2) and (3), $\frac{k}{ki} = \frac{Pm-QA}{Po-Pm}$,

$$\text{or, } Q = \frac{ki+k}{A\ ki} Pm - \frac{k\ Po}{A\ ki} \tag{6}$$

Pii

From (6), when Q = O,

$$Pm = Pii = \frac{k\ Po}{ki+k} \tag{7}$$

Pmi

At the end of inspiration, when Q = Qo, from (6)

$$Pm = Pmi = \frac{ki\ QoA}{ki+k} + \frac{k\ Po}{ki+k} \tag{8}$$

ti

From (6), $\frac{dQ}{dt} = \frac{ki+k}{A\ ki} \frac{d\ Pm}{dt}$

From (2), $\frac{dQ}{dt} = \frac{1}{ki^{1/2}} \sqrt{Po-Pm}$

Equating: $\frac{ki+k}{A\ ki} \frac{d\ Pm}{dt} = \frac{1}{ki^{1/2}} \sqrt{Po-Pm}$

Re-arranging and integrating:

$$\frac{ki+k}{A\ ki^{1/2}} \int_{Pii}^{Pmi} \frac{d\ Pm}{\sqrt{Po-Pm}} = t$$

$$ti = \frac{2(ki+k)}{A\ ki^{1/2}} \left(\sqrt{Po-Pii} - \sqrt{Po-Pmi}\right) \tag{9}$$

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EXPIRATION

Q and Pm

From (4) and (5), $\frac{k}{ke} = \frac{QA-Pm}{Pm-aPo}$

$$Q = \frac{ke+k}{Ake} Pm - \frac{ak}{Ake} Po \qquad (10)$$

Pix

At the start of expiration, let Q = Qo. At this point, Pm = Pix, and from (10),

$$Pm = Pix = \frac{Ake}{ke+k} Qo + \frac{ak}{ke+k} Po \qquad (11)$$

Pmx

At the end of expiration, when Q = O, from (10),

$$Pm = Pmx = \frac{ak}{ke+k} Po \qquad (12)$$

tx

From (10), $\frac{dQ}{dt} = \frac{ke+k}{Ake} \frac{dPm}{dt}$

From (5), $\frac{dQ}{dt} = \frac{-1}{ke^{1/2}} \sqrt{Pm-aPo}$

Equating: $\frac{ke+k}{Ake} \frac{dPm}{dt} = - \frac{1}{ke^{1/2}} \sqrt{Pm-aPo}$

Re-arranging and integrating:

$$\frac{ke+k}{Ake^{1/2}} \int_{Pix}^{Pmx} \frac{dPm}{\sqrt{Pm-aPo}} = -t$$

$$tx = \frac{2(ke+k)}{Ake^{1/2}} \left(\sqrt{Pix-aPo} - \sqrt{Pmx-aPo} \right) \qquad (13)$$

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COMMENT

The equations developed above allow the description of a complete cycle of the respirator within the limitations previously discussed. In addition, for simplicity, the further restriction is imposed that cycling begin and end at the relaxation volume of the lung (Q = O). Po, A, k, ke, and ki are given. In actual

practice, the mask pressure range is varied as desired, and Q, or the tidal volume, is thereby determined. As the present analysis is set up, Q is chosen, and this determines the cycling pressures. Other variables such as cycling time, total minute ventilation, effective minute ventilation, and flow rates are also dependent variables, and their values can be determined from the equations. Table 3 illustrates the calculated behavior of some of these variables with changing conditions. In this case, Po, the line pressure, is varied. A and k are chosen to be within the normal range. The inspiratory resistance is set at "Slow". In particular, the table indicates that a very considerable difference may exist between inspiratory and expiratory times. This theoretical prediction is well borne out by the real performance of the Valve on model lungs and on live subjects.

TABLE 3.
CALCULATED BEHAVIOR OF THE RESPIRATOR ON "SLOW" SETTING AT DIFFERENT LINE PRESSURES, NORMAL ADULT SUBJECT.

Po (cm. H_2O)	Q (liters)	Pmi (cm.H_2O)	Pii (cm.H_2O)	ti (seconds)	Pix (cm.H_2O)	Pmx (cm.H_2O)	tx (seconds)	P-range (cm.H_2O)	F/Min. (cycles/min.)	$\dot{V}$ (liters/min.)	Effective $\dot{V}$ (liters/min.)
90	.250	3.7	0.5	.59	2.2	-1.0	.42	4.7	59	14.8	3.0
	.500	7.4	0.5	1.25	5.5	-1.0	.79	8.4	29.4	14.7	8.8
	1.000	14.7	0.5	2.71	12.0	-1.0	1.42	15.7	14.5	14.5	11.6
	1.500	22.0	0.5	4.16	18.5	-1.0	1.94	23.0	9.8	14.7	12.8
	2.000	29.4	0.5	5.8	25.0	-1.0	2.42	30.4	7.3	14.6	13.1
53	.250	3.7	0.3	0.80	2.6	-0.6	.53	4.3	45	11.2	2.2
	.500	7.4	0.3	1.70	5.9	-0.6	.95	8.0	22.6	11.3	6.8
	1.000	14.7	0.3	3.64	12.4	-0.6	1.64	15.3	11.4	11.4	9.1
	1.500	22.0	0.3	5.82	18.9	-0.6	2.21	22.6	7.5	11.2	9.8
	2.000	29.4	0.3	8.3	25.4	-0.6	2.71	30.0	5.5	11.0	9.8
38	.250	3.7	0.2	1.01	2.8	-0.4	.58	4.1	37.8	9.5	1.9
	.500	7.4	0.2	2.12	6.1	-0.4	1.05	7.8	19.0	9.5	5.7
	1.000	14.7	0.2	4.58	12.6	-0.4	1.77	15.1	9.5	9.5	7.6
	1.500	22.0	0.2	7.46	19.1	-0.4	2.35	22.4	6.1	9.2	7.9
	2.000	29.4	0.2	11.2	25.6	-0.4	2.86	29.8	4.3	8.6	7.7
27	.250	3.7	0.1	1.21	2.9	-0.3	.66	4.0	32.0	8.0	1.6
	.500	7.4	0.1	2.64	6.2	-0.3	1.14	7.7	15.9	8.0	4.8
	.750	11.0	0.1	4.13	9.4	-0.3	1.54	11.3	10.6	8.0	5.8
	1.000	14.7	0.1	5.82	12.7	-0.3	1.90	15.0	7.8	7.8	6.2
	1.500	22.0	0.1	10.2	19.2	-0.3	2.50	22.3	4.7	7.1	6.1
20	.250	3.7	0.1	1.46	3.0	-0.2	.73	3.9	27.4	6.9	1.4
	.500	7.4	0.1	3.16	6.3	-0.2	1.24	7.6	13.6	6.8	4.1
	.750	11.0	0.1	5.06	9.5	-0.2	1.65	11.2	8.9	6.7	4.9
	1.000	14.7	0.1	7.50	12.8	-0.2	2.02	14.9	6.3	6.3	5.0

k = 3.5 cm. $H_2O/(liter/second)^2$
ki = 645 cm. $H_2O/(liter/second)^2$ (Inspiratory resistance: "Slow")
ke = 27 cm. $H_2O/(liter/second)^2$
A = 14.7 cm. H_2O/liter
Dead space = 200 ml.

SOME APPLICATIONS OF THE ANALYSIS

Change in respirator variables

The respirator variables are mask pressure range, line pressure, and inspiratory resistance. Changing the value of these changes the ventilation delivered by the respirator. Figure 3 shows the effect of changing these variables on the calculated total ventilation rate of a normal subject. It is apparent that total ventilation rate is increased by increasing the line pressure or decreasing the respirator resistance, but it is substantially independent of change in mask pressure range (cycling pressure range). This result is different from that obtained by Radford (1) in that ventilation volume does not fall off with an increase in mask pressure range. Figure 4 shows the results obtained with a simulated lung consisting of a tank with a volume of 68 liters and an orifice with a resistance of 3.2 cm. $H_2O/(liter/sec.)^2$. It is apparent that ventilation is altered by a change in line pressure alone but not by a change in mask pressure range alone. The results are in reasonable agreement with the calculated values of Figure 3. Although changing the mask pressure range does not alter total ventilation it greatly changes effective ventilation. At the lower mask pressures tidal volume is too low to provide much useful ventilation even though the rate is high. Increasing the pressure range increases tidal volume and effective ventilation. Figures 5 and 6 repeat the data of Figure 3, but this time in terms of effective ventilation.

Change in patient variables

For present purposes, the patient variables of primary importance are the compliance of the chest and lungs and the resistance.

Compliance of the lungs may easily be reduced to a third or a fourth of the normal value in persons with pulmonary disease or congestive heart failure. Disorders of the chest wall may greatly reduce compliance. Further, it is apparent that bronchial obstruction may reduce compliance simply by reducing the amount of lung which is available to change volume in response to a pressure change. According to the present analysis the total ventilation furnished by the resuscitator is independent of compliance. This emerges from an inspection of the equations. Equation (6) shows that the tidal volume, Q, is proportional to the compliance, 1/A. The times of inspiration (equation 9) and expiration (equation 13) are also proportional to the compliance. In calculating minute ventilation as tidal volume times cycles per minute, the factor of compliance cancels out. This result is similar to that of Fenn, Otis, and Rahn for the Burns resuscitator (4) and of Radford (1) for the general low pressure regulator. It is apparent, however, that a decrease in compliance can reduce effective ventilation by reducing the tidal volume toward ineffective levels. The influence of compliance changes on effective ventilation can readily be calculated from the equations presented above.

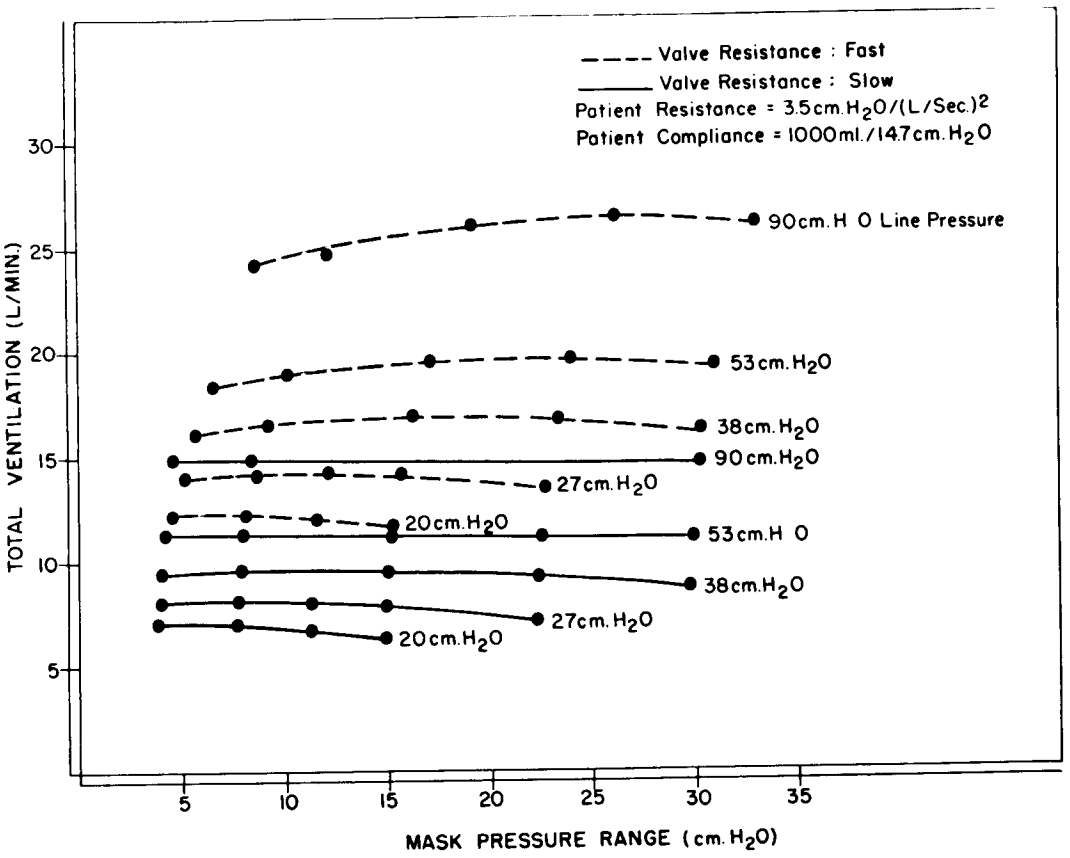

Figure 3. Calculated values of total minute ventilation at "Fast" and "Slow" resistances, a range of line pressures, and at different mask pressure ranges in a subject with normal compliance and a resistance slightly greater than normal.

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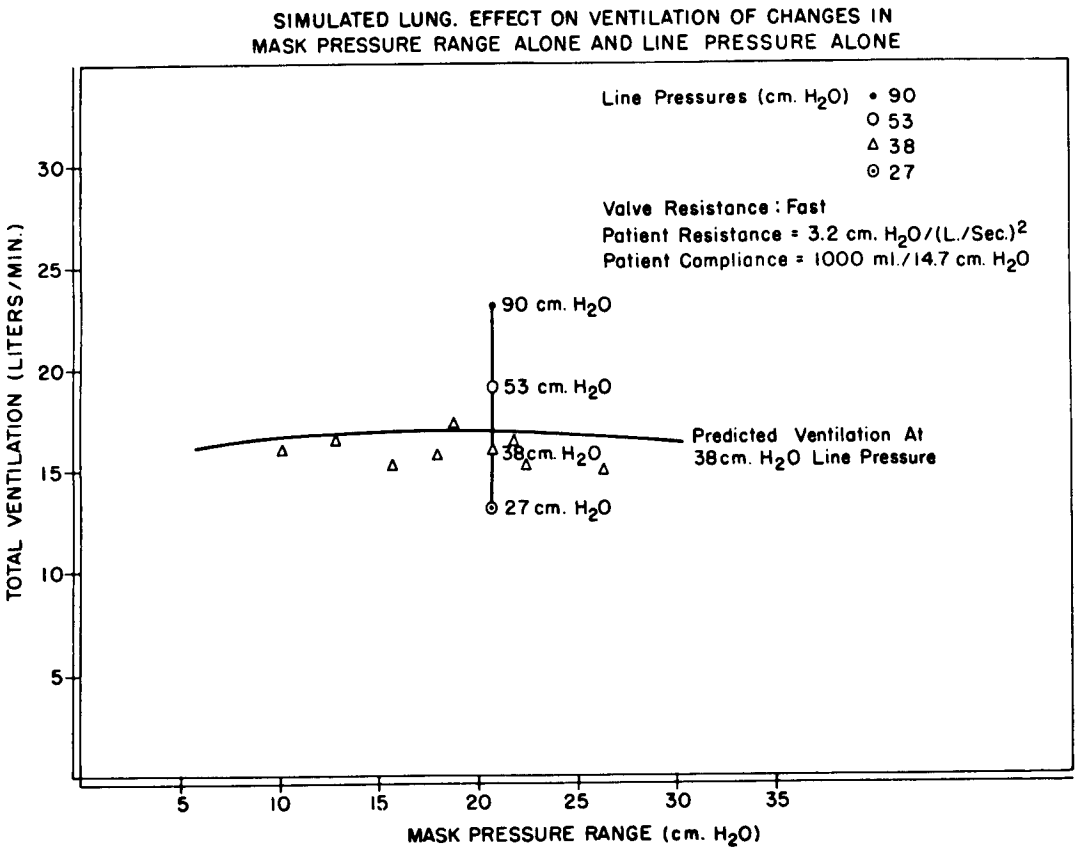

Figure 4. Actual values of minute ventilation obtained with the use of the Seeler resuscitator on a simulated lung. Ventilation rates with a line pressure of 38 cm. H_2O are close to the predicted values, indicated by the curved horizontal line. Variation in mask pressure range at a given line pressure has no significant effect on the total ventilation. Varying the line pressure at a constant mask pressure range changes ventilation markedly.

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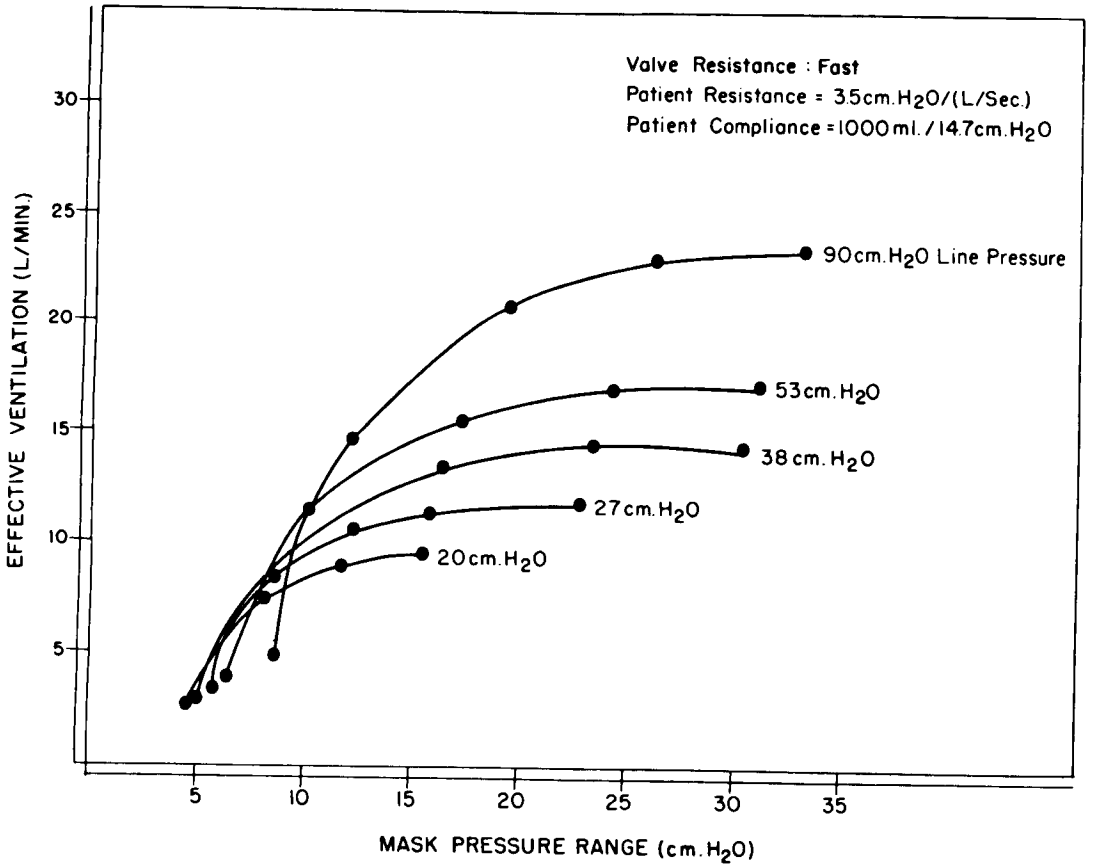

Figure 5. Calculated effective ventilation in a normal subject at different line pressures and "Fast" resistance. Respiratory dead space is assumed to be 200 ml. There is considerable dependence of effective ventilation on mask pressure range below pressure ranges of 15 to 20 cm. H_2O.

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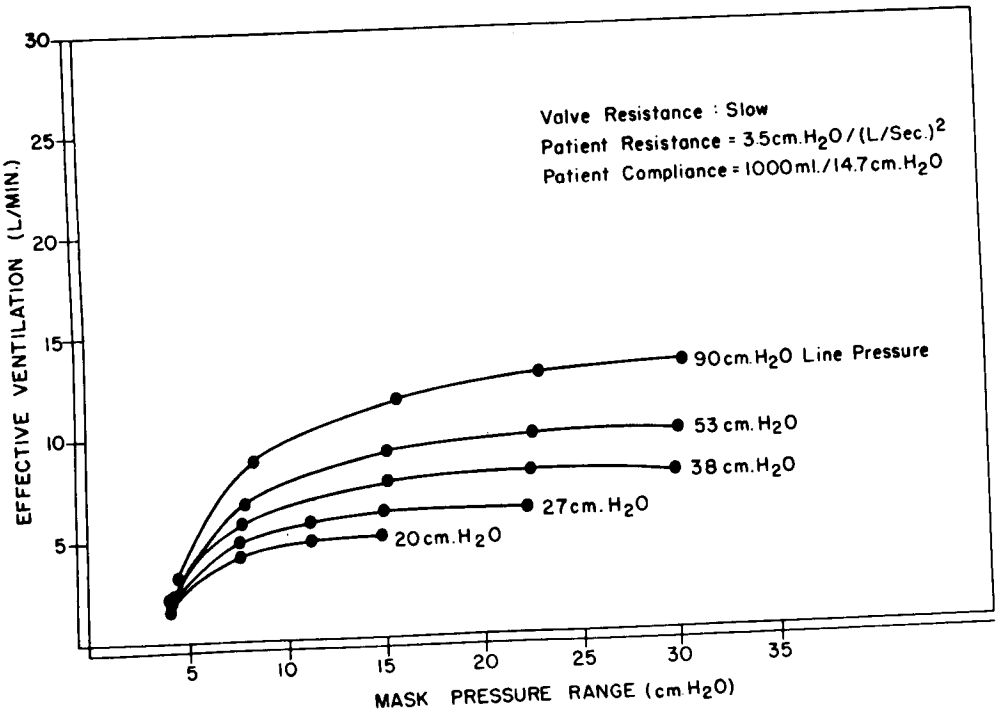

Figure 6. Calculated effective ventilation in a normal subject. Circumstances are the same as in Figure 5, except that the inspiratory resistance of the resuscitator is set at "Slow".

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On a general medical service increase in resistance is a much more important cause of disabling ventilatory insufficiency than is reduction in compliance. This was the case in the patients described elsewhere in this report who were treated with the resuscitator for carbon dioxide narcosis. For this reason and because the anti-cholinesterase compounds of military significance cause a large increase in respiratory resistance, particular attention has been directed toward the effect of a high patient resistance on resuscitator function.

A moderate increase in patient resistance, without change in other variables, causes a moderate decrease in total ventilation. This is illustrated by Figure 7, which shows the data of Figure 3 re-calculated for a person having a resistance of 32, rather than 3.5, cm. $H_2O/(liter/sec.)^2$. The effective ventilation is changed much more than the total ventilation by this increase in resistance. Figures 8 and 9 (which correspond to Figures 5 and 6) show the effective ventilation obtained against the increased patient resistance at the "Fast" and "Slow" settings of the resuscitator. No values are plotted for the highest line pressure (90 cm. H_2O) at the low Valve resistance ("Fast"). This is because effective ventilation under these conditions requires a mask pressure range over 35 cm. H_2O. Comparison of Figures 5 and 8 shows that a considerably greater mask pressure range is needed to begin effective ventilation at the "Fast" setting when the patient resistance is high than when it is low. At the "Slow" setting effective ventilation is much less altered by an increase in patient resistance. (See Figures 6 and 9).

In qualitative terms, an increase in patient resistance requires that the mask pressure be higher than before to maintain a given inspiratory flow and lower than before for a given expiratory flow. In actual practice it is desirable to avoid extreme mask pressures so far as possible in order not to damage portions of the lung which have less than the average resistance. To clarify the situation quantitatively, it is useful to re-examine the factors which determine Pii, that is the mask pressure developed at the start of the inspiratory cycle. According to Equation (7),

$$Pii = \frac{k\ Po}{ki+k} .$$

From this it appears that a high initial mask pressure is favored by a high line pressure and by a patient resistance which is high in relation to the inspiratory resistance of the resuscitator. Pii is independent of compliance. The effect on initial mask pressure of changing patient and respirator resistances without changing line pressure is illustrated in Table 4. From Table 4 it is apparent that initial mask pressure increases rapidly with an increase in patient resistance when the resuscitator resistance is low (85 cm. $H_2O/(liter/sec.)^2$). When Pii begins to approach closely the highest mask pressure which it is permissible to obtain, the tidal volume becomes reduced below the effective level. This is evident from re-arranging Equation (8) to show mask pressure, Pm,

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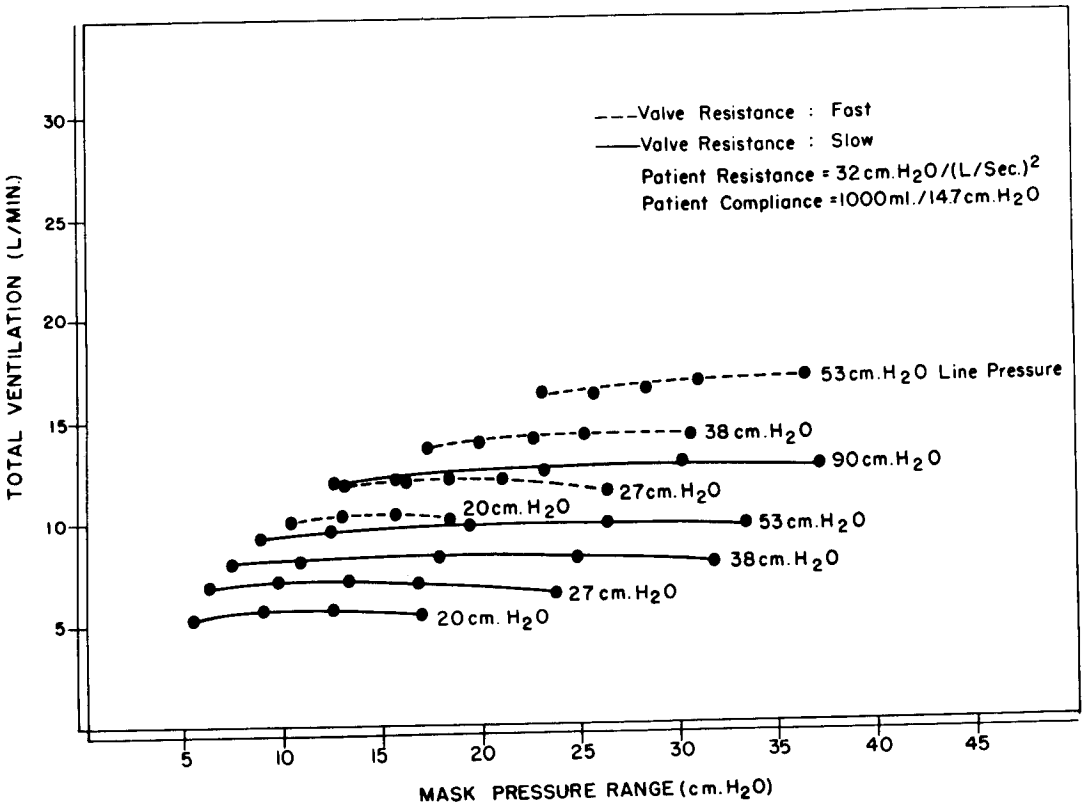

Figure 7. Calculated values of total minute ventilation at "Fast" and "Slow" resistances, a range of line pressures, and at different mask pressure ranges in a subject with normal compliance and much increased resistance. Compare Figure 3.

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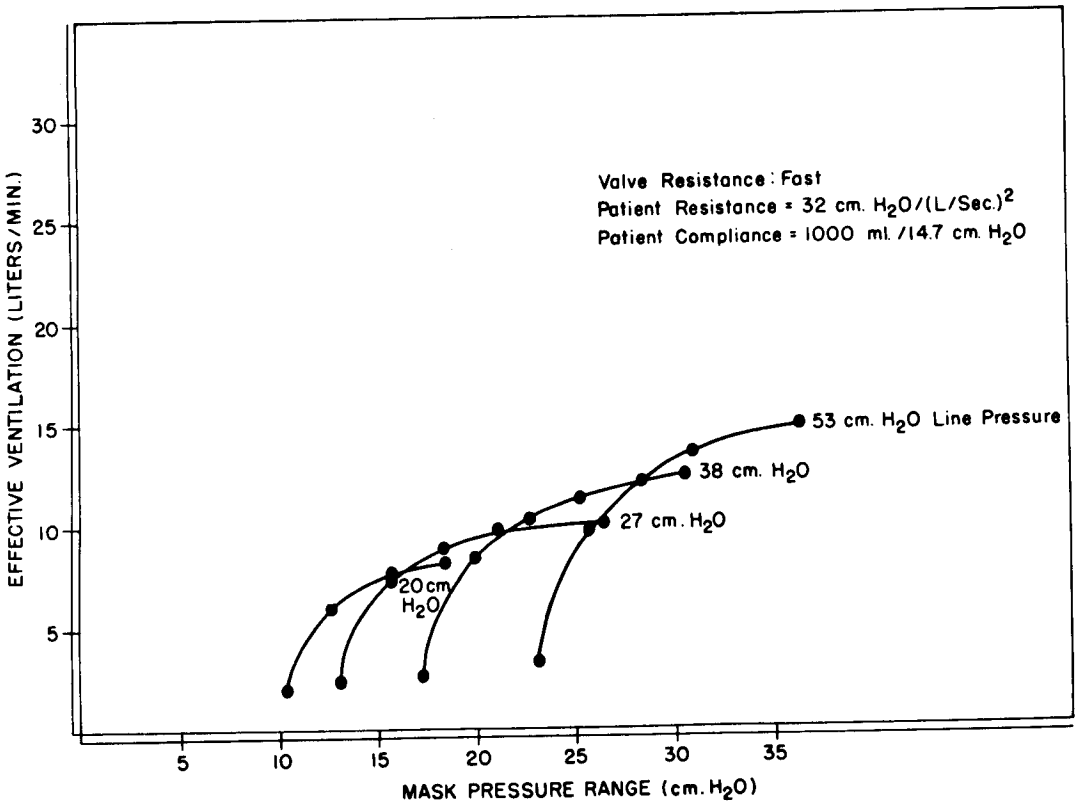

Figure 8. Calculated effective ventilation in a subject with increased resistance. Inspiratory resuscitator resistance set at "Fast". Compare Figure 5.

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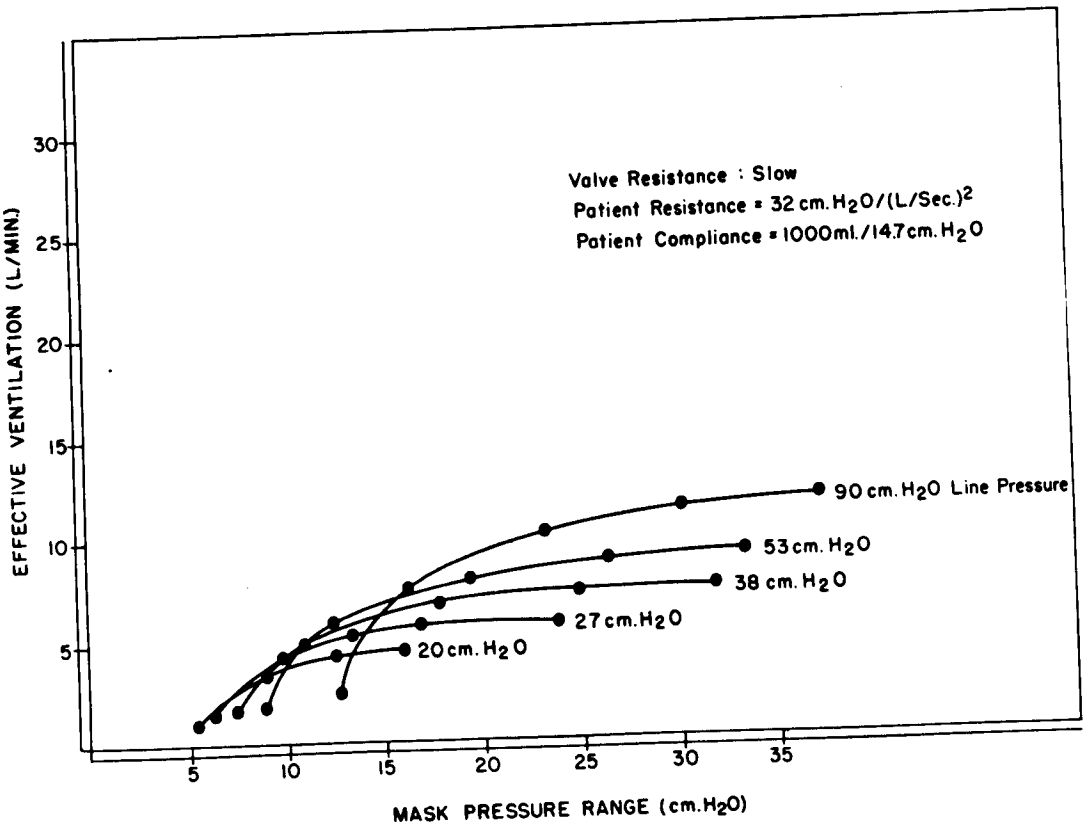

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Figure 9. Calculated effective ventilation in a subject with increased resistance. Resuscitator resistance set at "Slow". Compare Figure 6.

as a function of Pii and tidal volume, Q:

$$Pm = Pii + \frac{ki\ \Delta Q}{ki+k}$$

As Pii approaches Pm, Q must approach zero. When Pii exceeds the positive pressure at which the Valve is set to cycle, chattering will occur. If the cycling pressure can be set no higher, Pii must be reduced if any ventilation is to be provided.

TABLE 4.

THE EFFECT ON INITIAL INSPIRATORY MASK PRESSURE (Pii) OF CHANGING PATIENT RESISTANCE (k) AND INSPIRATORY RESUSCITATOR RESISTANCE (ki) AT A LINE PRESSURE OF 90 CM. H_2O.

	ki = 85 cm.H_2O/(liter/sec.)^2						
	k (cm.H_2O/(liter/sec.)^2)						
	0	1.7	3.5	9.9	16	25	32
Pii (cm. H_2O)	0	1.8	3.6	9.5	14.3	20.5	24.6

	ki = 645 cm.H_2O/(liter/sec.)^2						
	k (cm.H_2O/(liter/sec.)^2)						
	0	1.7	3.5	9.9	16	25	32
Pii (cm.H_2O)	0	0.2	0.5	1.4	2.2	3.4	4.3

Pii can be reduced by reducing the line pressure or by increasing the inspiratory resistance of the resuscitator (Table 4).

Combining these findings with information previously obtained about line pressure and valve resistance leads to the generalization that increasing line pressure and decreasing valve resistance usually tends to increase total ventilation but can reduce effective ventilation, depending upon the resistance of the patient. This suggests that there should be optimum values of line pressure and valve resistance for providing effective ventilation to a patient with a high resistance. For exploring this possibility a case has been worked out in which it is postulated that the patient has a resistance of 64 cm. H_2O/(liter/sec.)^2, A is 14.7 cm. H_2O/liter, and the mask pressure range is 30 cm. H_2O. With the line pressure held at 53 cm. H_2O, an examination is made of the effect of changing valve resistance; and with the valve resistance held at 85 cm. H_2O/(liter/sec.)^2, an examination is made of the effect of changing line pressure. The results are presented below.

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Changing inspiratory valve resistance alone

To calculate the effect on ventilation of changing valve resistance alone, it is necessary to make some approximations to cover the expiratory behavior of the valve at settings between the "Fast" and "Slow" resistances. The mask pressure at the end of expiration is given by Equation (12):

$$Pmx = \frac{a\,k}{ke+k}\,Po.$$

Under the given conditions, this yields a Pmx of -8.8 cm. H_2O at the "Fast" setting and -3.7 cm. H_2O at "Slow". It is assumed that there is a linear relationship between inspiratory valve resistance and Pmx in the interval between these two points. Pmi, the mask pressure at the end of inspiration, is next obtained from the relation:

$$Pmi = 30 + Pmx,$$

since a pressure range of 30 cm. H_2O has been postulated. Q is obtained from Equation (8), Pii from (7), and ti from (9). For tx, the expiratory time, a further approximation is needed. The ratio of tx to ti is determined for the "Fast" and "Slow" settings and it is assumed that a linear relationship holds between this ratio and the valve resistance in the interval between these two settings. This allows calculation of the total cycling time. Since the tidal volume is known, the total ventilation and effective ventilation can be calculated for any value of inspiratory resistance between the "Fast" and "Slow" settings.

The results of these calculations for the particular case postulated above are presented in Figure 10. Under these circumstances it is apparent that the optimum valve resistance is close to 200 cm. $H_2O/(liter/sec.)^2$. As a practical matter, however, effective ventilation does not fall off rapidly as valve resistance is further increased. When the patient resistance is decreased by half, the effective ventilation continues to increase as the resuscitator resistance is reduced to its lowest setting.

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Changing line pressure alone

In this case, the "Fast" resuscitator resistance (85 cm. $H_2O/(liter/sec)^2$) is arbitrarily chosen, and the line pressure is reduced. Calculations are made as usual, and the results are presented in Figure 11. It is apparent that the maximum effective ventilation occurs at a line pressure of about 35 cm. H_2O.

The example demonstrates the existence of an optimum value for resuscitator resistance alone at a given line pressure and for line pressure alone at a given resuscitator resistance in providing effective ventilation to a patient with a high resistance. It is apparent that an optimum for the two values together could also be found. However, any extensive analysis of this kind, using the present methods, would require the services of a computer.

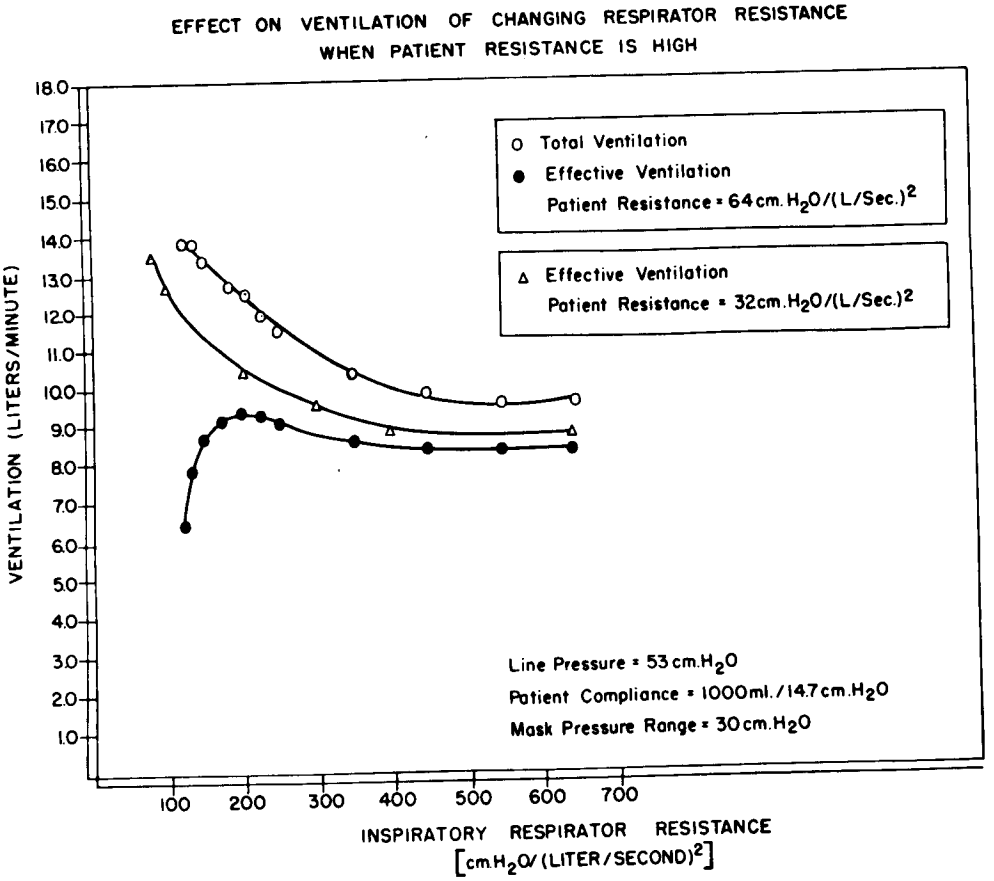

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Figure 10. The effect on total and effective ventilation of changing resuscitator resistance when the patient resistance is high. Line pressure is held constant. It is apparent that there is a maximum for effective ventilation at a resistance near 200 cm. $H_2O/(liter/second)^2$.

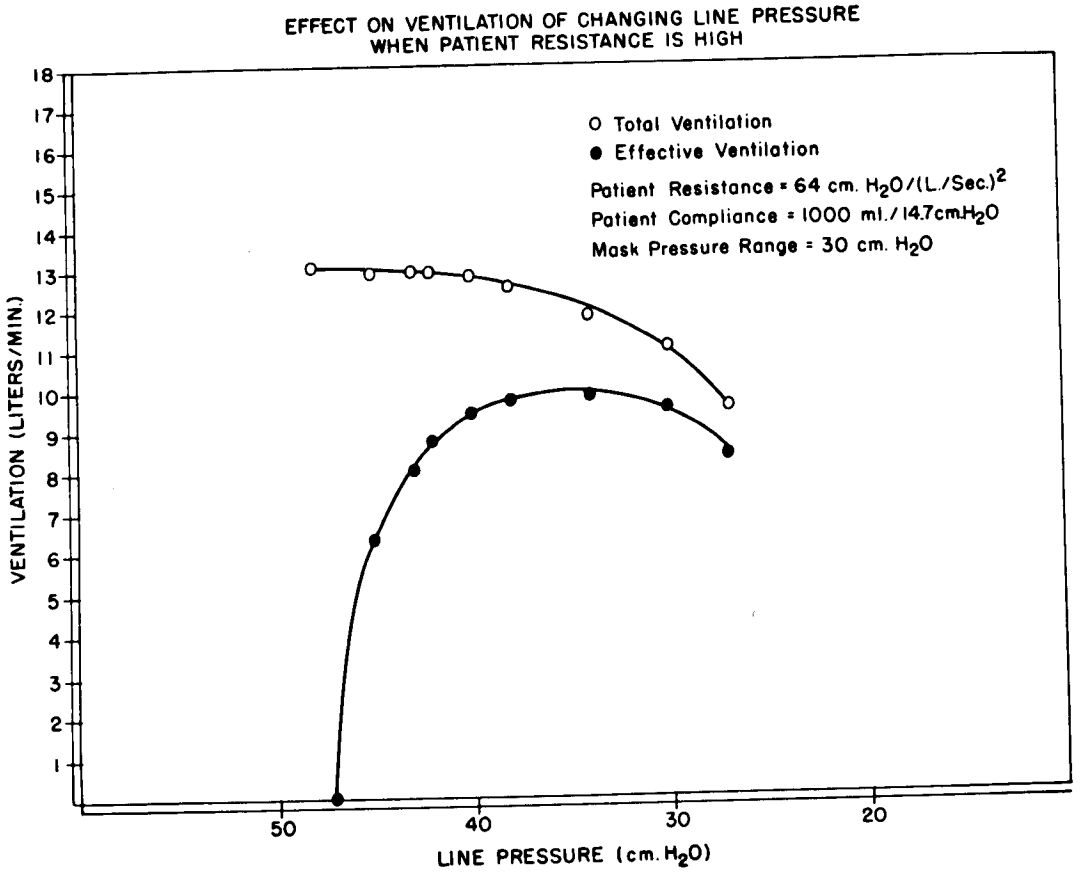

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Figure 11. The effect on total and effective ventilation of changing line pressure when the patient resistance is high. Resuscitator resistance is held constant.

Comment

From the results of this analysis some general observations can be made on the subject of providing adequate ventilation to a patient with a high resistance and a low compliance. It is obviously useful for this purpose to have as large a range of mask pressure as possible. The upper limit of 37 cm. H_2O positive pressure set by the "Dill Committee" (5) is considerably greater than the 22 cm. H_2O furnished by the Seeler resuscitator model used in this study. For providing ventilation to subjects with a high resistance, the negative phase is not very effective, as pointed out before. In order to realize the full advantages of permissible mask pressure the Seeler resuscitator for hospital use should allow mask pressures up to 37 cm. H_2O. During inspiration, the mask pressure is related to line pressure, resistances, compliance, and tidal volume as shown by Equation (8):

$$Pm = \frac{ki\ QA}{ki+k} + \frac{k\ Po}{ki+k}$$

The last expression on the right is equal to Pii (Equation 7), the mask pressure developed at the onset of inspiration. If this exceeds the mask pressure at which the valve is set to cycle, the valve will "chatter". Pii may be reduced by lowering line pressure or increasing resuscitator resistance. This will leave more of the mask pressure available for increasing the tidal volume Q. It will also, however, reduce the flow rate (Equation 2) and thereby increase the cycling time. The first action will tend to increase effective ventilation and the second will tend to reduce it. As indicated above, an optimum value of line pressure or resuscitator resistance for providing effective ventilation can be found in a given set of circumstances.

The question now arises whether it is useful to be able to vary both line pressure and resuscitator resistance, or whether one variable alone, in addition to mask pressure range, would be sufficient to adjust the resuscitator to changing needs. From a practical viewpoint, the resistance should be made variable, since it is easy to provide for this, while a variable line pressure may not always be available. If the line pressure must be fixed, it should be somewhat in excess of the 37 cm. H_2O pressure recommended as the upper limit of mask pressure. However, this line pressure may provide greater effective ventilation or more rapid cycling than desired if the valve resistance is turned to minimum to obtain the greatest sucking effect from the venturi. At lower line pressures, if they are made available, a good sucking effect can still be obtained at a lower flow rate (see Figure 2, "Fast" setting). It is evident that the resuscitator can be well controlled without varying the line pressure, but that having this capability provides a more exact control.

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Solution for the case where $P = k\dot{V}$

If it is assumed that there is a linear relationship between pressure and gas flow in the subject ($P = k\dot{V}$), as postulated by Radford (1), the basic equations have a somewhat different form:

$$Pp = QA \tag{a}$$

For inflow through the respirator:

$$\left(\frac{dQ}{dt}\right)^2 ki = Po - Pm \tag{b}$$

For inflow into the patient:

$$\frac{dQ}{dt}\, k = Pm - QA \tag{c}$$

For outflow from the patient:

$$-\frac{dQ}{dt}\, k = QA - Pm \tag{d}$$

For outflow through the respirator:

$$\left(\frac{dQ}{dt}\right)^2 ke = Pm - a\ Po \tag{e}$$

or

$$\frac{dQ}{dt} = -\frac{1}{ke^{1/2}}\sqrt{Pm - a\ Po}$$

INSPIRATION

Q and Pm

From (b) and (c),

$$Q = \frac{1}{A}\left(Pm - \frac{k}{ki^{1/2}}\sqrt{Po - Pm}\right) \tag{f}$$

Pii

From (f), when $Q = 0$,

$$Pii = \frac{k}{2ki}\left(\sqrt{k^2 + 4ki\ Po} - k\right) \tag{g}$$

ti

From (b),

$$\frac{dQ}{dt} = \frac{1}{ki^{1/2}}\sqrt{Po - Pm}$$

Rearranging (f) and differentiating with respect to t,

$$\frac{dPm}{dt} = -\frac{k}{2ki^{1/2}\sqrt{Po - Pm}}\left(\frac{dPm}{dt}\right) + A\,\frac{dQ}{dt}$$

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Substituting for $\frac{dQ}{dt}$ the value given above, rearranging, and integrating,

$$ti = \frac{ki^{1/2}}{A} \int_{Pii}^{Pmi} \frac{d\,Pm}{\sqrt{Po-Pm}} + \frac{k}{2A} \int_{Pii}^{Pmi} \frac{d\,Pm}{Po-Pm}$$

$$ti = \frac{2ki^{1/2}}{A} \left(\sqrt{Po-Pii} - \sqrt{Po-Pmi}\right) + \frac{k}{2A} \ln \frac{Po-Pii}{Po-Pmi} \quad (h)$$

EXPIRATION

Q and Pm

From (d) and (e),

$$-\frac{dQ}{dt} = \frac{\sqrt{Pm - a\,Po}}{ke^{1/2}} = \frac{QA - Pm}{k}$$

Solving for Pm,

$$Pm = QA + \frac{k}{2ke} \left(k - \sqrt{k^2 + 4ke\,(QA - a\,Po)}\right) \quad (i)$$

Pix

This is obtained from (i), letting Pm = Pix when Q = Qo.

Pmx

At the end of expiration, when Q = 0,
From (i)

$$Pmx = \frac{k}{2ke} \left(k - \sqrt{k^2 - 4ke\,a\,Po}\right) \quad (j)$$

tx

From (d) and (e),

$$Pm = QA - \frac{k}{ke^{1/2}} \sqrt{Pm - a\,Po}$$

Differentiating with respect to t, and substituting the value of $\frac{dQ}{dt}$ obtained from (e),

$$\frac{dPm}{dt} = -\frac{A}{ke^{1/2}} \sqrt{Pm - a\,Po} - \frac{k}{2ke^{1/2}\sqrt{dPm - a\,Po}} \left(\frac{dPm}{dt}\right)$$

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Re-arranging and integrating,

$$tx = -\frac{ke^{1/2}}{A}\int_{Pix}^{Pmx}\frac{dPm}{\sqrt{Pm - a\,Po}} - \frac{k}{2A}\int_{Pix}^{Pmx}\frac{dPm}{Pm - a\,Po}$$

$$tx = \frac{2ke^{1/2}}{A}\left(\sqrt{Pix - a\,Po} - \sqrt{Pmx - a\,Po}\right) + \frac{k}{2A}\ln\frac{Pix - aPo}{Pmx - aPo} \quad (k)$$

The equations presented immediately above which describe inspiration are the same as those given by Radford (1). Those describing expiration are peculiar to this analysis, since they are based in part on experimental data pertaining to the Seeler Valve. This system of equations embodies the assumption that there is a linear relationship between pressure and gas flow in the subject ($P=k\dot{V}$). The system of equations on which the present analysis is based assumes that the relationship has the form, $P=k\dot{V}^2$. Our data indicate that the latter expression holds better than the former for subjects with a high resistance. When the resistance of the subject is not abnormally high, it makes little difference which relationship is chosen. Tables 5A and 5B demonstrate the close quantitative similarity of the ventilation patterns yielded by

TABLE 5A

VENTILATION CALCULATED ON BASIS THAT $P=k\dot{V}$ FOR SUBJECT'S AIR FLOW.
$P=3.5$ cm. H_2O when $\dot{V}=1$ liter/sec. Compliance = 1000 ml/14.7 cm.H_2O.
$Po=53$ cm.H_2O. $ki=85$ cm.H_2O/(L/sec.)^2. $ke=64$ cm.H_2O/(L/sec.)^2.

Q(liters)	Pmi (Cm.H_2O)	Pii (cm.H_2O)	ti (seconds)	Pix (cm.H_2O)	Pmx (cm.H_2O)	tx(sec.)	P range (cm.H_2O)	Cycles per sec.	$\dot{V}$(L/min.)	Effective $\dot{V}$(L/min.)	$\dot{V}$ if ti=tx
.201	5.6	2.9	.25	1.1	-1.7	.38	7.3	95	19.2	0.1	23.8
.510	10.0	2.9	.69	5.4	-1.7	.93	11.7	37	18.9	11.5	22.2
.930	16.0	2.9	1.30	11.4	-1.7	1.59	17.7	20.8	19.3	15.2	21.4
1.420	23.0	2.9	2.08	18.3	-1.7	2.27	24.7	13.8	19.6	16.9	20.5
1.920	30.0	2.9	2.96	25.4	-1.7	2.92	31.7	10.2	19.6	17.5	19.5

calculations based on these two separate pressure-flow relationships in a subject whose resistance is low. The assumption is made that a pressure of 3.5 cm. H_2O produces an air flow of 1 liter per second, that compliance is 1000 ml/14.7 cm. H_2O, line pressure

TABLE 5B

VENTILATION CALCULATED ON BASIS THAT $P=k\dot{V}^2$ FOR SUBJECT'S AIR FLOW.
P=3.5 cm.H_2O when $\dot{V}$=1 liter/sec. Compliance = 1000 ml/14.7 cm.H_2O.
Po=53 cm.H_2O. ki=85 cm.H_2O/(L/sec.)^2. ke=64 cm.H_2O/(L/sec.)^2.

Q(liters)	Pmi (cm.H_2O)	Pii (cm.H_2O)	ti (seconds)	Pix (cm.H_2O)	Pmx (cm.H_2O)	tx (seconds)	P range (cm.H_2O)	Cycles per sec.	$\dot{V}$(L/min.)	Effective $\dot{V}$(L/min.)
.250	5.6	2.1	.34	2.6	-0.9	.48	6.5	73	18.3	3.7
.500	9.2	2.1	.69	6.1	-0.9	.90	10.1	38	18.9	11.4
1.000	16.2	2.1	1.42	13.0	-0.9	1.67	17.1	19.4	19.4	15.5
1.500	23.2	2.1	2.22	19.9	-0.9	2.34	24.1	13.1	19.7	17.1
2.000	30.2	2.1	3.24	26.9	-0.9	2.98	31.1	9.6	19.2	17.3

is 53 cm.H_2O, and the inspiratory and expiratory resistances of the valve are those obtained at the "Fast" setting.

The total ventilation, calculated as described above, does not change significantly with the mask pressure range, using either of the above assumptions for the pressure-flow relationships of the patient. If, however, it is assumed, as in Radford's analysis (1), that the time of expiration equals that of inspiration, there is a progressive fall in total ventilation with increase in mask pressure range, as indicated in the last column of Table 5A. This fall did not actually occur in studies on a simulated lung (Figure 4). With increasing valve resistances, there is an increasing difference between ventilation rates calculated as described in this report and those calculated on the assumption that expiratory time equals inspiratory time. As pointed out before, this assumption of equal inspiratory and expiratory times does not in general apply well to the actual behavior of the Seeler valve.

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SUMMARY

1. Equations have been developed by means of which the function of the Seeler resuscitator can be described within the limitations imposed by certain assumptions. The most restricting of the assumptions is that compliance and resistance of the subject remain constant throughout the respiratory cycle.

2. On the basis of these equations an examination has been made of the effect on ventilation of changing the resuscitator variables of

line pressure, mask pressure range, and inflow resistance, and of changing the compliance and resistance of the subject.

3. Total ventilation is increased by increasing line pressure or reducing inflow resistance, but is not altered by changing mask pressure. Effective ventilation is altered by changing mask pressure and falls rapidly as mask pressure is reduced to low values. These general conclusions are confirmed by data obtained from trials of the resuscitator on a simulated lung.

4. Total ventilation is independent of compliance, but effective ventilation is readily diminished by a decrease in compliance. A moderate increase in patient resistance causes only a moderate decrease in total ventilation, but the effective ventilation may be sharply reduced, the extent of this reduction depending upon the values of the other variables. The effect is much less when the resuscitator resistance is high than when it is low.

5. Particular attention has been devoted to the problem of providing effective ventilation to a patient with a high resistance. For this purpose it is important to have available as high a mask pressure and as large a mask pressure range as is consistent with reasonable safety. Increasing line pressure and lowering valve resistance will increase total ventilation but will have the effect of increasing mask pressure at the start of inspiration. Increasing mask pressure will ultimately lead to a reduction in effective ventilation. It is demonstrated that optimum values of line pressure and valve resistance can be found for providing effective ventilation in a particular case.

6. For realizing the full usefulness of the resuscitator in hospital use, it is recommended that both line pressure and inflow resistance be made variable.

7. The present analysis assumes that pressure-flow relationships for the subject take the form $P=k\dot{V}^2$, where airway resistance is high. An alternate analysis is developed for the case that $P=k\dot{V}$, as assumed in a previous study. It is demonstrated that substantially identical results are obtained with the two expressions for the ventilation of a subject with a normal resistance.

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SECTION II

USE OF THE SEELER RESUSCITATOR IN THE TREATMENT OF CARBON DIOXIDE NARCOSIS

INTRODUCTION

It is now well recognized (6,7,8) that carbon dioxide narcosis can occur in persons with disorders that greatly reduce alveolar ventilation. During the last four years the authors have made observations on 22 examples of this condition. As a rule, these patients have had pulmonary emphysema or diffuse pulmonary fibrosis with marked ventilatory impairment. On this background, acute ventilatory insufficiency has usually developed through one of two basic mechanisms: Ventilation may become mechanically more difficult, particularly as a result of respiratory infections; or the respiratory center may be depressed, as by anesthetics or sedative drugs. Occasionally these patients lapse into coma without an apparent precipitating cause. In persons with chronic ventilatory impairment carbon dioxide loses most of its effectiveness as a respiratory stimulant, but in high concentrations it still has a narcotic effect. The narcotic effect is usually not manifest until arterial carbon dioxide tensions in the region of 80 mm. Hg, or higher, have been attained. Since an air-breathing person near sea level has somewhat less than 150 mm. Hg available in his alveoli for the partial pressures of oxygen and carbon dioxide together, these high alveolar tensions of carbon dioxide necessitate low alveolar oxygen tensions. Because of this and the underlying lung disease, the arterial blood oxygen is low in these people, and most of their ventilatory drive is provided by the peripheral chemo-receptors which respond to low arterial oxygen tensions. In this situation a further increase in arterial carbon dioxide tension stimulates respiration mostly by way of the attendant decrease in oxygen tension. Consequently, severe carbon dioxide narcosis does not often develop while these people are breathing air; if it does develop, the subject is in danger primarily from the associated oxygen deprivation. When oxygen is administered, as it often must be, the situation is changed. With a high arterial blood oxygen tension, the ventilatory effort is markedly reduced, and the carbon dioxide tension rapidly reaches narcotic levels. The danger now arises from the depressant effects of high carbon dioxide tensions and the resultant severe acidosis.

In carbon dioxide narcosis the therapeutic effort is directed toward increasing alveolar ventilation. We customarily make use of bronchoscopic drainage, bronchodilators, ACTH or hydrocortisone, and antibiotics. In addition, particularly with comatose patients, we have freely used mechanical respirators, including the Seeler resuscitator.

The successful use of a resuscitator in these subjects is difficult because of their high respiratory resistance and low

compliance. Similar problems would be encountered in resuscitating victims of "nerve gas" poisoning, and for this reason the performance of a resuscitator on persons with carbon dioxide narcosis is particularly significant in evaluating the device. Furthermore, some familiarity with the clinical problems of carbon dioxide narcosis should be useful to those who may have to treat cases of nerve gas poisoning with resuscitators. If a resuscitator can provide even very slight alveolar ventilation, and can do it with oxygen, then the primary respiratory problem is apt to be carbon dioxide retention, rather than oxygen lack.

It is the purpose of this report to describe the results obtained and the experience gained with the use of the Seeler resuscitator in four patients with carbon dioxide narcosis.

METHODS

The Seeler resuscitator used in these studies was a special model designed to permit relatively high flow rates. Its characteristics have been described in detail elsewhere in this report.

Ventilation was measured with a wire screen flow meter. Pressure drops across the screen, mask pressure, line pressure, and arterial pressure were measured with Statham strain gages and photographically recorded. Arterial blood oxygen was determined by a photometric method (9); pH by a glass electrode using Rosenthal's factor for temperature correction (10); and blood carbon dioxide content by the manometric method of Van Slyke and Neill (11). Blood carbon dioxide tensions were calculated from these data in the usual way. Use of these methods on blood samples equilibrated with known high carbon dioxide tensions indicates that calculation of "indirect" pCO_2 is satisfactory up to CO_2 tensions of at least 150 mm. Hg.

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RESULTS

Four persons with carbon dioxide retention have been treated with the Seeler resuscitator. Data on these patients are presented below. In addition, two patients who were conscious resisted the use of the resuscitator to the extent that it could not be employed successfully. In one subject (Case 4) sufficient data were obtained to allow a comparison between the actual performance of the patient-resuscitator system and that predicted by the equations developed elsewhere in this report.

Case 1: W.P., a 74 year old business man, was admitted unconscious with the diagnosis of a cerebral vascular accident. For many years he had had exertional dyspnea, and fifteen years before admission a diagnosis of pulmonary emphysema had been made. Three years before admission studies had shown marked ventilatory impair-

ment but nearly normal arterial blood oxygen, carbon dioxide, and pH. For a week before admission he had felt weak but had continued work and had driven home from the office on the day before admission. On the day he came to the hospital he had felt very weak and had remained in bed. Over a period of about thirty minutes on this day his weakness progressed markedly, he became disoriented, and shortly thereafter he lapsed into coma. On admission, he had a blood pressure of 140/70 mm. Hg, a heart rate of 120, and a respiratory rate of 30 to 60 per minute. The skin was cyanotic. The heart had a diastolic gallop rhythm. There were occasional movements of all extremities but there was no response to painful stimuli. The pupils were small and reacted sluggishly to light. Tendon reflexes and superficial abdominal reflexes could not be obtained. The Babinski response was not present. During the first two hospital days he had fever, with a maximum temperature of 39.5° C on the first day. The leukocyte count on admission was 14,600. Lumbar puncture showed a cerebrospinal fluid pressure of 300 mm. H_2O, but the findings were otherwise normal.

Oxygen was administered, with disappearance of the cyanosis but no other change. Arterial blood studies (see table 1) showed a high carbon dioxide tension and severe acidosis. An endotracheal tube was inserted and was left in place for the next fifteen hours. Brief trials of the Seeler resuscitator showed that it was quite successful in reducing the carbon dioxide tension. Accordingly, the resuscitator was used for the next fifteen hours, on a fifteen minute on - fifteen minute off schedule. When the resuscitator was not being used, nasal oxygen was given. ACTH was administered. On the morning of the second hospital day he was greatly improved and was responding well to commands. Arterial pH was back to normal (Table 1). Thereafter he made a steady improvement and was discharged on the 14th hospital day. After consciousness was fully regained an attempt was made on one occasion to assist his breathing with the Seeler resuscitator. He found this very disagreeable and after 5-10 breaths he refused to try further.

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On the basis of our experience with this condition before the employment of vigorous therapy, we believe that this patient would have died without the use of the resuscitator. It is noteworthy that the resuscitator functioned well on the patient when he was comatose, but he would not permit its use after he became alert. During recovery the arterial pH rose slightly above normal although the pCO_2 was still abnormally high. This is a common finding in these patients during recovery while their alkaline reserve is still falling. In one patient in our series an arterial pH of 7.48 with a pCO_2 of 47 mm. Hg, was found during spontaneous breathing in the recovery period. It is probable that this mild alkalosis results from a returning sensitivity of the respiratory center to carbon dioxide tension, so that ventilation is forced to the point of a moderate elevation in pH. In this changing situation, the blood picture is somewhat like that of a metabolic alkalosis which is being partially compensated by carbon dioxide retention. The use of ACTH in this transient state of hypochloremic alkalosis, during which it is difficult to give potassium by mouth, suggests

TABLE 1.

CARBON DIOXIDE NARCOSIS IN A PATIENT WITH PULMONARY EMPHYSEMA.
Use of Seeler Resuscitator.

Comment	Arterial pH	Arterial CO_2 Tension (mm. Hg)
Spontaneous breathing, on oxygen.	6.98	168
After 15 minutes on Seeler resuscitator.	7.27	70
After 8 minutes of spontaneous breathing.	7.16	
30 minutes after last use of resuscitator.	7.07	117
After 20 minutes on Seeler resuscitator.	7.30	68
Spontaneous breathing after overnight use of the Seeler resuscitator.	7.43	54

that careful attention be paid to serum potassium levels. To date, however, we have had no instances of hypochloremic, hypokalemic alkalosis during ACTH therapy in these patients.

Case 2: A.D., a 65 year old woman, had had emphysema, recurrent attacks of bronchitis, and hypertension for over ten years. A year before the present admission she had been hospitalized in a semicomatose condition following sedation with barbiturates. Four to five days before admission she began to have more severe dyspnea than usual and a worsening of her cough. On admission she demonstrated moderately severe dyspnea at rest, inspiratory and expiratory rhonchi, a blood pressure of 180/100, and cardiac enlargement, but no clear evidence of congestive heart failure. During the evening of the first day she was given 3 grams of chloral hydrate in two separate doses and managed to add to this sedation by consuming some whiskey which she had brought in her luggage. It was noted that she was in a deep sleep. The following morning she was found to be comatose and cyanotic. A consultant was called, and he arrived with the Seeler resuscitator just as respirations ceased. Resuscitation was begun. Twenty minutes later the first arterial blood sample was obtained. The findings on this and later samples are presented in Table 2. After several hours on the resuscitator, she began to regain consciousness, resuscitation was discontinued, and she resumed spontaneous respiration. Recovery was uneventful and she was discharged on the twelfth hospital day.

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TABLE 2.

CARBON DIOXIDE NARCOSIS IN A PATIENT WITH EMPHYSEMA AND OVER-SEDATION.
Use of the Seeler Resuscitator.

Comment	Arterial O_2 percent Saturation	Arterial pH	Arterial CO_2 Tension (mm.Hg)
After use of the resuscitator for 20 minutes. Line pressure 33 cm.H_2O. Mask pressure plus 8 to minus 8 cm.H_2O. Resistance intermediate. Tank oxygen.	99.5	7.18	106
After resuscitation for one hour; now using compressed air.	53.5	7.31	70
Several hours later, patient breathing spontaneously. On nasal oxygen.	82.6	7.26	81

Case 3: M.P., a 40 year old housewife had had asthma for 22 years and was admitted to the hospital in a severe attack. The attack became progressively more severe and by the seventh hospital day she was cyanotic and disoriented. Bronchoscopic drainage was carried out and ACTH was started. Arterial blood studies during spontaneous breathing and with the use of resuscitators were carried out, and these are detailed in Table 3. The first two samples, taken while the patient was breathing spontaneously, illustrate the increase in pCO_2 which may occur in these people when arterial hypoxia is relieved by breathing oxygen. Use of the Bennett resuscitator kept the patient well oxygenated and prevented an increase in pCO_2. The Seeler resuscitator appeared to provide somewhat better ventilation than the Bennett in this patient under the conditions of use, judging from the arterial pCO_2 values. However, the patient accepted the Bennett resuscitator much more readily than the Seeler resuscitator, presumably because of the high peak flow rate of the former. During the next two days the Bennett resuscitator was used occasionally. After the second day no ventilatory assistance was needed, and she made an uneventful recovery.

This case illustrates the problem that may be posed by a disoriented but still conscious patient whose ventilation is failing but who can still resist the use of a resuscitator. We have had the opportunity of seeing in consultation two patients, one with kyphoscoliosis and pulmonary fibrosis, and the other

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TABLE 3.

EARLY CARBON DIOXIDE NARCOSIS IN A PATIENT WITH SEVERE ASTHMA.
Use of two different resuscitators.

Comment	Arterial O_2 percent Saturation	Arterial pH	Arterial CO_2 Tension (mm.Hg)
Spontaneous breathing, air	77.2	7.19	80
Spontaneous breathing, oxygen	94.2	7.15	96
Four hours later, off oxygen and on air for 5 minutes.		7.18	84
On Bennett resuscitator, 5 minutes. Mask pressure plus 24 to 0 cm. H_2O.	98.6	7.23	76
Spontaneous breathing, air off O_2 for 7 minutes	82.8	7.26	68
On Seeler resuscitator, 5 minutes. Mask pressure plus 16 to minus 8 cm. H_2O.	99.2	7.29	62
Two days later, spontaneous breathing, air	95.3	7.47	41

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with emphysema and bronchitis, who resisted the use of both Seeler and Bennett resuscitators to the extent that neither apparatus was useful. Both patients collapsed and died with unexpected rapidity and never entered a prolonged stage of carbon dioxide narcosis. We have had similar experiences with the Drinker respirator. This problem does not arise in persons who are heavily sedated by their own carbon dioxide. Subjects who have had carbon dioxide narcosis for several hours do not recover immediately when their pCO_2 is decreased by resuscitation, so that a resuscitator can usually be employed without difficulty for many hours. When the pCO_2 has been reduced substantially for a prolonged period, these subjects do not easily lapse into narcosis again. In addition, prolonged use of a resuscitator allows time for ACTH and antibiotics to have some effect in clearing the airway. Until recently we have hesitated to sedate patients who were still conscious and making some respiratory effort of their own, because we were not certain of improving on the ventilation which the patient was providing for himself. Recently we have seen in consultation two patients with severe carbon dioxide

retention who were resisting ventilation by the Drinker respirator. Heavy sedation with demerol in one case and morphine in the other eliminated struggling and allowed much more effective ventilation. Both patients recovered. In the future we expect to use sedation freely to avoid the problem of patient resistance. Case 2 is an example of the inadvertent use of sedation to produce a situation which was easily managed with the aid of the Seeler resuscitator.

Case 4: R.T., a 58 year old negro male, was known to have hypertension but had otherwise been well until he had a hemorrhage in the pons. He lived three days but never regained consciousness. On admission to the hospital, the first day of his illness, he was having obvious respiratory difficulty. For this reason a tracheal tube was inserted. Respiration was very shallow. Large quantities of mucus were produced and repeated aspirations were required. Blood pressure on admission was 240/130 mm. Hg, but it soon declined, and from the first day a continuous infusion of norepinephrine was required to maintain an adequate blood pressure. On the second hospital day respiration became progressively more irregular and feeble. Arterial blood measurements at this point and thereafter are presented in Table 4. For the next 48 hours, until death from circulatory failure, respiration was maintained quite satisfactorily with the Seeler resuscitator. Despite

TABLE 4.

ARTERIAL BLOOD GASES OF PATIENT WITH STROKE.
EFFECT OF USING SEELER RESUSCITATOR OVER A 48 HOUR PERIOD.

Comment	Arterial O_2 percent Saturation	Arterial pH	Arterial CO_2 Tension (mm.Hg)
Second hospital day, breathing spontaneously, shortly after stopping nasal O_2.	68.8	6.98	149
Seeler valve, line pressure 24 cm.H_2O. Mask pressure plus 13 to minus 2 cm.H_2O, "Fast".	99.9	6.98	149
Line pressure 90, mask pressure plus 24 to minus 11, resistance setting intermediate.	100.0	7.17	85
Two hours later. Line pressure 53, mask pressure plus 23 to minus 10, resistance intermediate.	99.3	7.34	50
Third day. Valve settings as above.	100.0	7.33	53

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correction of the respiratory acidosis there was no significant improvement, but none was to be expected in view of the pontine hemorrhage subsequently found at autopsy.

From these findings it appears quite likely that CO_2 narcosis may well appear in persons with head injuries or illnesses associated with respiratory depression and prolonged unconsciousness, and its occurrence may seriously jeopardize the chances of recovery.

The use of nor-epinephrine deserves some comment. The marked instability of blood pressure exhibited by this patient probably resulted from damage to the hind brain. However, we have seen large falls in blood pressure during resuscitation of other persons with carbon dioxide narcosis. In two cases who recovered a nor-epinephrine infusion had to be maintained almost continuously for more than twenty-four hours.

COMPARISON OF ACTUAL WITH PREDICTED PERFORMANCE OF THE RESUSCITATOR IN A SUBJECT WITH HIGH RESISTANCE AND LOW COMPLIANCE.

In one of the subjects, R.T., who had a tracheal airway, it was possible to make enough measurements to determine to what extent the theory described in Section I might predict the behavior of the patient-resuscitator system. The compliance and resistance of the patient were calculated from recordings of mask pressure and ventilation according to the method described by Radford (12). The calculations were modified to conform with the present assumption that patient resistance follows the rule: $P = k\dot{V}^2$. Data for the calculations were obtained at a line pressure of 53 cm. H_2O, mask pressure range of 32.4 cm. H_2O, and respirator resistance set at "Fast". End-inspiratory resistance was estimated at 21 cm. $H_2O/(liter/second)^2$ and end-expiratory resistance at 46 cm. $H_2O/(liter/second)^2$. The effective resistance was taken to be 33, the mean of the inspiratory and expiratory resistances, and this value was used in the calculation of compliance. Compliance was estimated to be 1000 ml/35 cm. H_2O. Using these values for resistance and compliance and the line pressures and mask pressure ranges actually used on the patient, calculations of the predicted behavior of the patient-resuscitator system were made from the equations of Section I. The results for three different conditions are presented in Table 5. There is a general correspondence between "actual" and "predicted" values, but the agreement is not particularly close. The best agreement occurs at the highest line pressure. This is because the values for patient compliance and resistance were calculated from data obtained under these circumstances. The calculated resistance and compliance of this patient changed with each change in the operating conditions of the resuscitator. In general, mean patient resistance decreased as the mask pressure was increased. This result indicates that a high mask pressure has an advantage in providing effective ventilation beyond that predicted in the theoretical analysis, i.e., decrease in patient resistance. The analysis does not take account of changes

TABLE 5.

COMPARISON OF THEORETICALLY PREDICTED WITH ACTUAL VENTILATION PROVIDED BY SEELER RESUSCITATOR.
Patient resistance, 33 cm.H_2O/(liter/second)^2; compliance, 1000 ml./35 cm.H_2O.

	Line pressure (cm.H_2O)	Respiratory Resistance	Mask Pressure Range(cm.H_2O)	Tidal Volume (liters)	Insp. Time (seconds)	Exp. Time (seconds)	Resp. Rate (Cycles/min.)	Total Vent. (L/Min.)	Effective Ventilation (L/Min.)
Predicted	53	"F"	32.4	.460	.74	.86	37.5	17.2	9.6
Actual	53	"F"	32.4	.430	.85	.96	33.3	14.2	7.7
Predicted	27	"F"	17.2	.264	.60	.72	45.4	12.0	2.9
Actual	27	"F"	17.2	.183	.55	.56	54.0	9.9	0.0
Predicted	24	"S"	15.3	.390	2.40	1.08	17.1	6.7	3.3
Actual	24	"S"	15.3	.210	1.37	.67	29.4	6.2	.3

in properties of the patient which may result from the operation of the resuscitator.

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SUMMARY

1. The use of the Seeler resuscitator in four cases of carbon dioxide retention is described. In two cases, the resuscitator appeared to be responsible for the patient's recovery. In two additional cases voluntary resistance by the patient precluded the use of the resuscitator.

2. Some clinical problems associated with the resuscitation and general care of persons with carbon dioxide narcosis are discussed as they arise in connection with the cases reported here.

3. In one case a detailed comparison is made between the ventilation provided by the resuscitator under different operating conditions and that predicted on the basis of the resistance and compliance of the patient. There is a general, but not close, agreement between actual and predicted ventilation. Discrepancies are partly the result of changes in the patient's resistance in response to changes in operating conditions of the resuscitator.

4. It is concluded that the Seeler resuscitator has been very useful in the treatment of carbon dioxide narcosis.

SECTION III

SOME OBSERVATIONS ON THE EFFECTS OF INDUCING HYPOCAPNIA DURING MECHANICAL RESUSCITATION.

INTRODUCTION

A resuscitator which has the capability for providing adequate ventilation in a subject with lung disease can easily produce hypocapnia in a person with a normal respiratory system. There is some indication that the development of hypocapnia during resuscitation may have undesirable consequences in persons with certain types of disorders. These are disorders in which there is a danger of accentuating peripheral circulatory collapse or cerebral hypoxia. In addition, transient hyperkalemia may occur during acute hypocapnia to an extent which may present a hazard in persons predisposed to cardiac arrhythmias.

It is well known that hypocapnia induced by hyperventilation causes a fall in the arterial blood pressure of normal persons. There is evidence that hypocapnia acts directly on the blood vessels to produce a net vasodilatation and that this is responsible for the fall in pressure (13). This net vasodilatation is the resultant of widely different local effects, such as vasoconstriction in the skin and brain and vasodilatation in the muscles and the splanchnic region. In normal subjects the vasodilatation is opposed and limited by the vasomotor reflexes which normally maintain arterial blood pressure. In persons with impaired function of the sympathetic nervous system the fall in blood pressure is much greater and more prolonged (13,14). This effect has been demonstrated in persons with "idiopathic" autonomic disease, in those who have had sympathectomies for hypertension, and after sizable doses of hexamethonium. The effect may be anticipated in persons given some other drugs which block autonomic function, and in spinal anesthesia. In electrolyte disturbances, such as sodium deprivation, the vasomotor response to a fall in blood pressure is often very deficient, and hypocapnia would be expected to produce a severe fall in blood pressure. In traumatic shock, either primary or secondary to blood loss, hypocapnia induced by resuscitation might further lower the blood pressure. Such a result could be catastrophic. It has been found that positive pressure breathing has a deleterious effect on animals in hemorrhagic shock. The conventional explanation that positive pressure obstructs venous return to the heart is probably correct. In the light of our present understanding of hypocapnia, however, it is highly desirable that such studies should include control of the arterial pCO_2. If increasing the mask pressure causes an increase in alveolar ventilation, the resultant hypocapnia might be responsible in part for the effect on blood pressure.

Hypocapnia has particular importance for the cerebral circulation since it constricts the cerebral vessels at the same time that it tends to lower the arterial pressure. This point has been stressed by Himwich (15). In consequence of these effects, hypo-

capnia reduces cerebral blood flow and lowers the oxygen tension of the brain. In normal persons this effect is not dangerous, but it could conceivably be dangerous in certain subjects who require resuscitation.

It has been reported that an acute elevation of arterial pCO_2 in experimental animals can cause marked hyperkalemia and that abrupt reduction of the pCO_2 to normal levels may cause a further transient rise in serum potassium (16). The changes in potassium concentration are large, and can be associated with profound electrocardiographic changes, including arrhythmias. It is apparent that this experimental situation might be simulated by a period of prolonged under-ventilation followed by over-ventilation, occurring during resuscitation or during surgical anesthesia with controlled respiration. In normal conscious subjects, voluntary hyperventilation can easily cause a transient increase of 20 to 50 percent in serum potassium concentration (17). In some subjects a sharp increase in serum potassium may have untoward consequences, particularly in the induction of cardiac arrhythmias.

Under certain circumstances the production of hypocapnia during resuscitation may have advantages. Many disoriented or semi-stuporous patients fight a mechanical resuscitator, presumably because the resuscitator cannot provide a peak flow rate which is fast enough to impress the patient as being appropriate to his need. If it is possible to produce mild hypocapnia, such subjects will often abandon respiratory efforts of their own and submit peaceably to the respirator. In effect, hypocapnia is a convenient respiratory sedative for such persons.

In neurosurgical practice, over-ventilation is sometimes used to reduce the blood volume of the brain, thus making it easier to handle during surgical procedures, as for example in the closure of a craniotomy. The cerebrospinal fluid pressure can be reduced markedly by hypocapnia. It seems likely that this effect, though relatively temporary, may have useful clinical applications, for example in the treatment of a medullary "pressure cone" or herniation into the foramen magnum, following lumbar puncture.

Many of these considerations are highly speculative. However, when they are taken together they indicate that much more information is needed about the effects of inducing hypocapnia during resuscitation in a variety of conditions.

It is the purpose of the present report to describe the effects on blood pressure and jugular blood oxygen of inducing hyperventilation in unconscious subjects by means of the Seeler resuscitator.

METHODS

Most of the pertinent methods have been described in

Section II. The resuscitator was supplied by air or oxygen through a regulator which could provide line pressures up to 90 cm. H_2O. During the hyperventilation experiments on normal subjects a line pressure of 90 cm. H_2O was used. The resuscitator resistance was set at "Fast" or as near this as it could be set without rapid cycling. Six subjects with normal cardiorespiratory systems were studied during hyperventilation under light pentothal anesthesia. One subject, a male of 51 with normal lungs, had apnea following the rupture of a basilar artery aneurysm. Ventilation was thereafter maintained with the Seeler resuscitator until death from circulatory failure 40 hours later. This subject, who had severe medullary injury, required a constant infusion of nor-epinephrine to maintain his blood pressure near its usual level.

RESULTS

Effect of hyperventilation on arterial pressure.

After a control period, during which observations were made in the six normal anesthetized subjects during spontaneous breathing, hyperventilation by the Seeler resuscitator was carried out for an average of 2.2 minutes. As shown in Figure 1, ventilation was sufficiently vigorous to produce a considerable decrease in arterial pCO_2 during this time. The mean mask pressures were near 12 to 14 cm. H_2O. Mean arterial pressures declined only very slightly in these normal, lightly anesthetized subjects. In the subsequent apneic period, which averaged 1.8 minutes, the mean arterial pressure showed little further change. A moderate fall in arterial pressure can be produced in normal persons during the more profound hypocapnia which can be achieved by voluntary hyperventilation (13).

A different result was obtained in the unconscious subject whose vasomotor regulation was impaired after a medullary hemorrhage. Figure 2 shows the course of his arterial pressure during hyperventilation on oxygen for 2 minutes at a line pressure of 53 cm. H_2O and a resistance setting of "Fast". The respiratory rate was 14 per minute and the ventilation was estimated from line pressure and resuscitator resistance to be about 19 liters per minute. Immediately prior to this a ventilation rate of 7 liters per minute had been maintained. During hyperventilation the mean mask pressure rose from 6 to 11 cm. H_2O, and it is quite possible that this change may have contributed to the considerable arterial pressure drop seen in Figure 1. However, the gradual course of the decline in pressure and its subsequent rise in the apneic period suggests that hypocapnia, rather than an abrupt change in mask pressure, was the major factor in these arterial pressure changes. Table 1 presents the effect of different resuscitator settings on arterial blood gases in this subject.

This effect of hypocapnia on blood pressure is similar to that which can be produced by voluntary hyperventilation in conscious persons who have impaired vasomotor regulation due to sympathetic dysfunction. Also, in such persons an elevation of

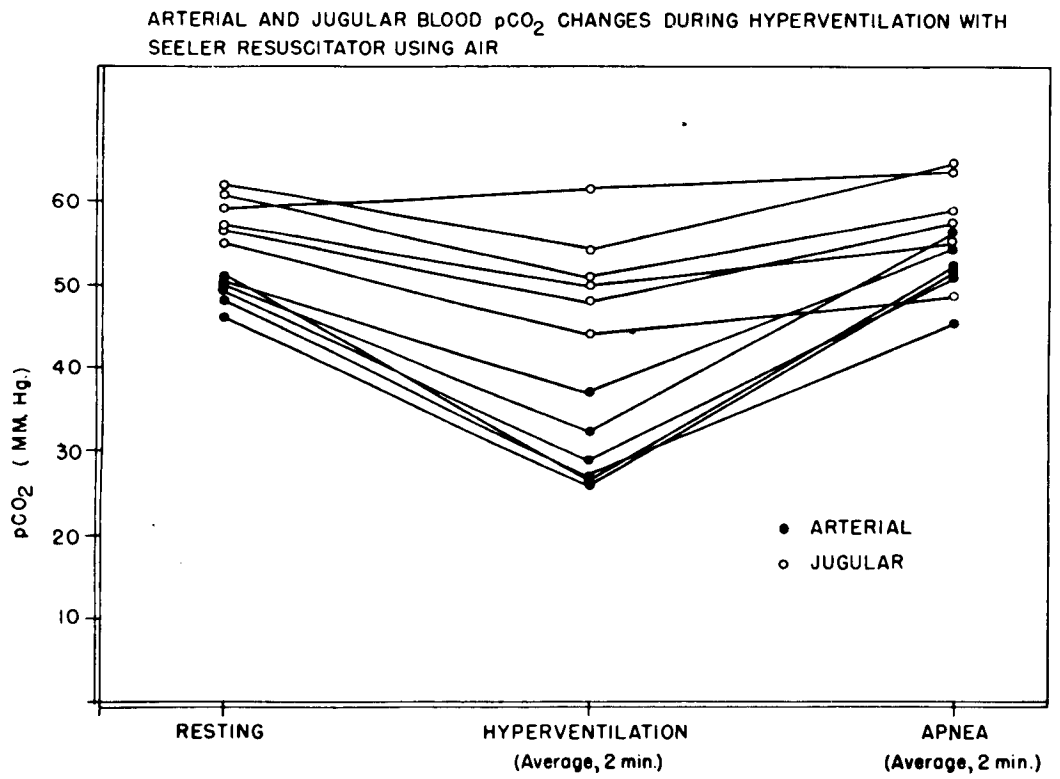

Figure 1. The figure shows the arterial and jugular pCO_2 levels during light pentothal anesthesia in six subjects in the resting state, during hyperventilation (using air) with the Seeler resuscitator at a rate of about 25 liters per minute, and at the end of the subsequent period of apnea. It is apparent that hyperventilation was sufficient to cause a considerable drop in arterial pCO_2, but that the jugular pCO_2 showed much less change.

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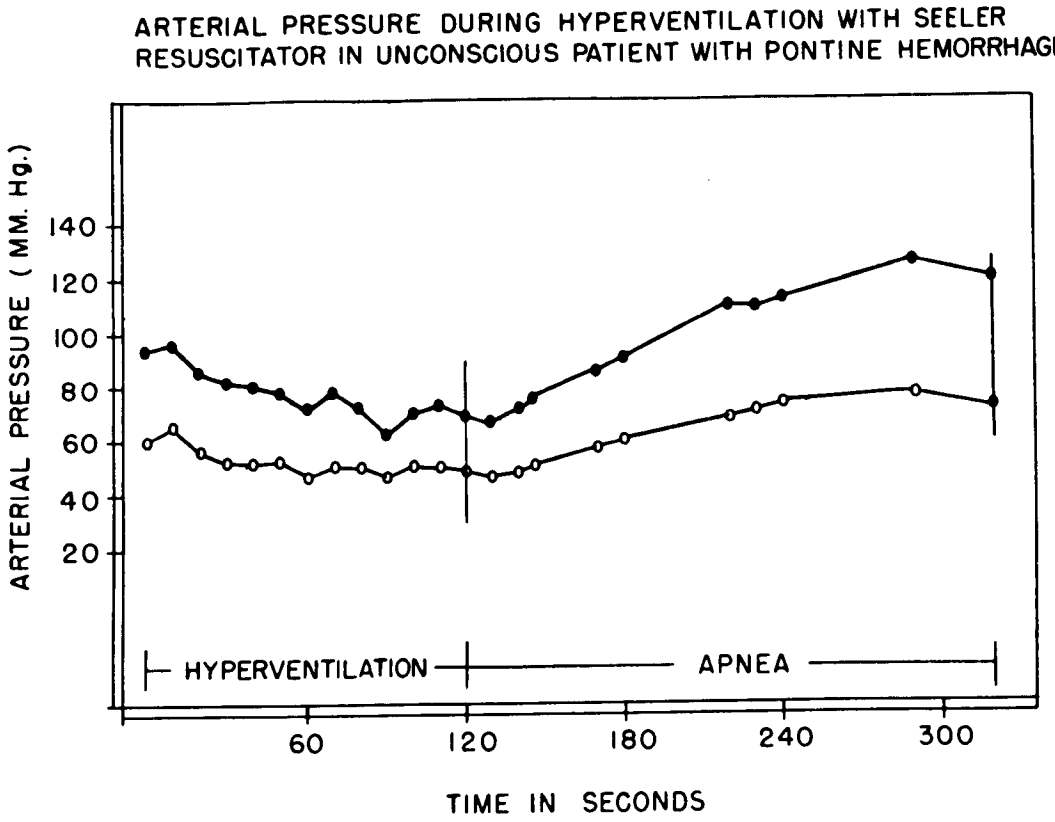

Figure 2. Effect of hyperventilation on arterial pressure (direct, femoral artery) of a 51 year old male who was apneic after rupture of a basilar artery aneurysm. Respiration was maintained by the Seeler resuscitator for 40 hours, until death from circulatory failure. Prior to the period of hyperventilation shown in the figure, the ventilation rate was 7 liters per minute, and during hyperventilation it was 19 liters per minute. The resuscitator was being supplied by tank oxygen. It is apparent that hyperventilation produced a progressive fall in arterial blood pressure and that the pressure rose to above control levels during a prolonged period of subsequent apnea.

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arterial pCO_2 above the normal resting level may cause a considerable increase in arterial blood pressure.

TABLE 1.

ARTERIAL BLOOD VALUES AT DIFFERENT RESUSCITATOR SETTINGS IN A SUBJECT WITH MEDULLARY HEMORRHAGE.

Comment	Arterial Blood pCO_2 Saturation	Arterial Blood pH	Arterial Blood pCO_2 (mm. Hg)
Line pressure 27 cm. H_2O Resistance near "Slow"	99.6	7.36	50
Line pressure 27 cm. H_2O Resistance intermediate	99.8	7.40	42
Line pressure 20 cm. H_2O Resistance "Fast"	99.9	7.43	36
Line pressure 53 cm. H_2O Resistance "Fast"	99.7	7.48	28

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Effect of hyperventilation on the composition of jugular venous blood in normal subjects anesthetized with pentothal.

The effect of hyperventilation on arterial and jugular venous pCO_2 is shown in Figure 1. As expected, there is a considerable decrease in arterial pCO_2 but much less change in the jugular blood. With regard to blood oxygen, this effect is reversed (Figure 3), in that there is a considerable alteration in jugular blood, but only a slight change in arterial blood. These effects depend upon the decrease in cerebral blood flow resulting from cerebral vascular constriction. As pointed out above, the decrease in jugular venous blood oxygen has no danger in a normal subject breathing air at sea level, but it could conceivably become dangerous under other circumstances.

As would be expected, oxygen breathing during hyperventilation increases somewhat the percent saturation of jugular venous blood over that which is found during hyperventilation on air. Some observations were made on the extent to which the use of oxygen could compensate for the decrease in cerebral blood flow induced by hyperventilation. Figure 4 shows the difference in each

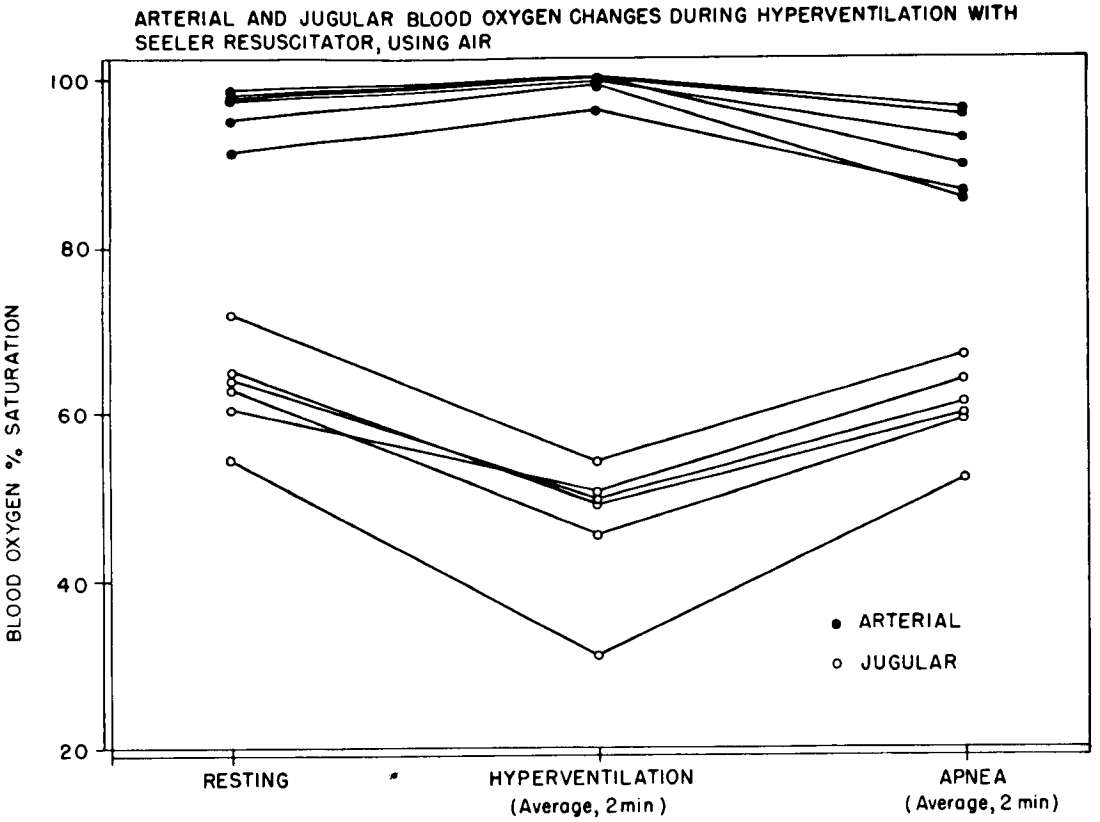

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Figure 3. Percent oxygen saturation of arterial and jugular venous blood of six subjects under light pentothal anesthesia at rest, during hyperventilation (using air) with the Seeler resuscitator at a rate of about 25 liters per minute, and at the end of the subsequent period of apnea. It is apparent that hyperventilation caused a considerable drop in the oxygen content of blood leaving the brain.

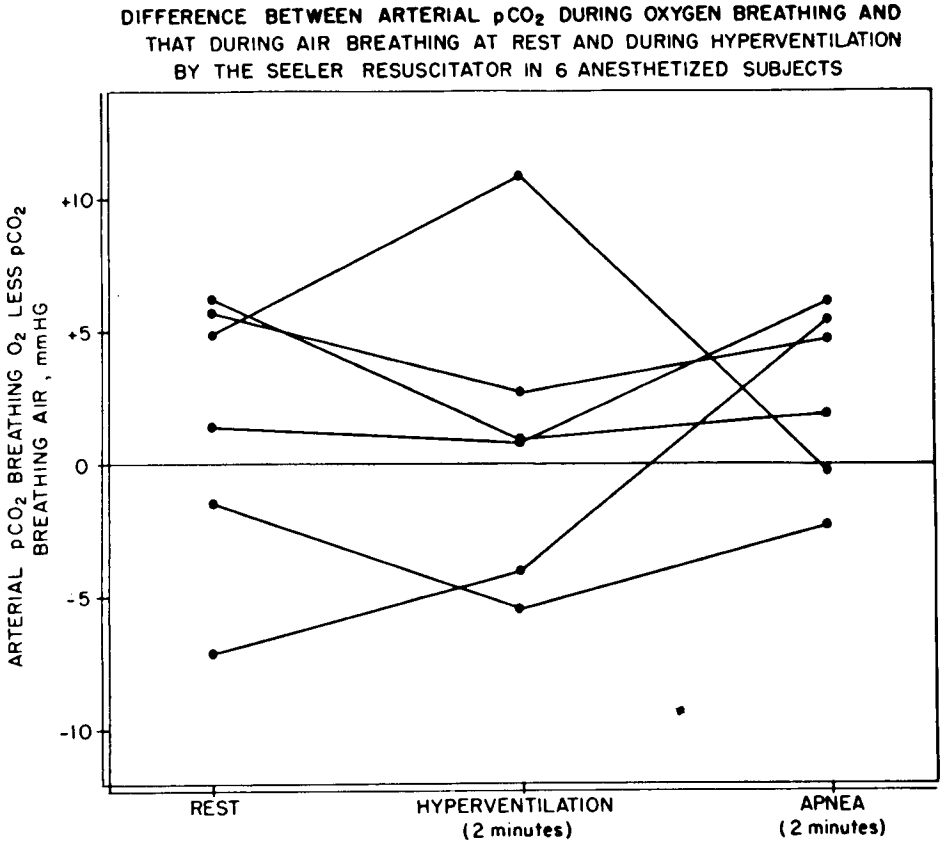

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Figure 4. Each point represents, for a given subject, the difference in arterial pCO_2 during oxygen breathing and that during air breathing. As expected, there is no consistent, overall difference between the pCO_2 values with the use of the two different gases at rest, during hyperventilation, or at the end of apnea. This result indicates that the hyperventilation on oxygen was mechanically comparable to that on air.

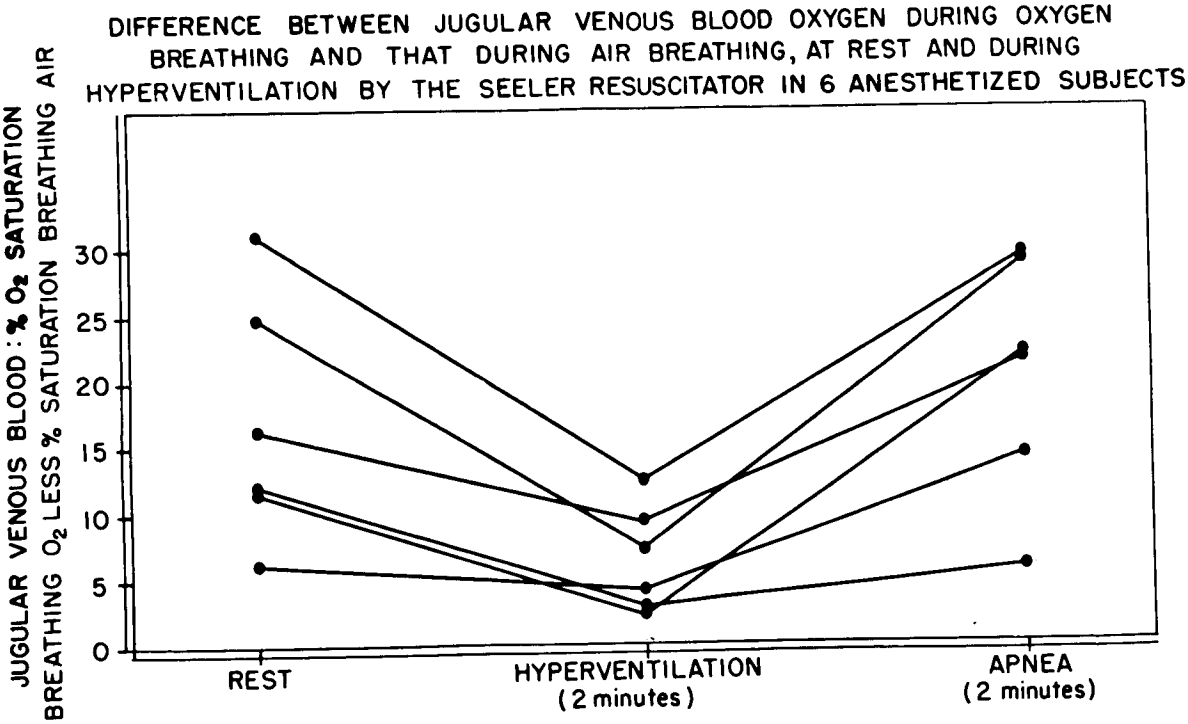

Figure 5. Each point represents, for a given subject, the difference between jugular venous blood oxygen saturation during oxygen breathing and that during air breathing. As expected, the jugular blood oxygen at rest is considerably increased by breathing oxygen. During hyperventilation, on oxygen, however, there is a very considerable drop in jugular oxygen, toward the levels found during hyperventilation on air. The decrease in jugular blood oxygen is greater during oxygen over-breathing than during air over-breathing. The reasons for this are not clear, but the finding indicates that oxygen breathing does not prevent the development of subnormal cerebral venous oxygen tensions during hyperventilation, although it does lessen the effect.

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subject between the arterial pCO_2 while on oxygen and that while breathing air, at rest, during hyperventilation, and at the end of the subsequent apneic period. Since the differences are not large under any of these circumstances, it is apparent that hyperventilation was about as vigorous while the subject was on oxygen as it was while he was on air. This result was, of course, to be expected, since the operating conditions of the resuscitator were the same under the two circumstances. Although hyperventilation was roughly equivalent on oxygen and air, as measured by arterial CO_2 wash-out, Figure 5 shows that the drop in jugular blood oxygen was greater during oxygen over-breathing than during air over-breathing. In some cases the jugular blood oxygen content during hyperventilation on oxygen came close to that during hyperventilation on air. The reasons for this are not clear, but the finding indicates that oxygen breathing does not prevent the development of subnormal cerebral venous oxygen tensions during hyperventilation, although it does lessen the effect.

SUMMARY

1. The Seeler resuscitator can easily produce hypocapnia in a subject with a normal respiratory system. The development of hypocapnia during resuscitation has potential hazards and, under special circumstances, may have some advantages.

2. In six normal subjects under light pentothal anesthesia there was no significant fall in blood pressure during hyperventilation with the resuscitator. In a patient with a medullary hemorrhage and defective vasomotor regulation brief hyperventilation produced a very significant fall in blood pressure.

3. Hyperventilation with the Seeler resuscitator can produce a very considerable drop in the oxygen content of cerebral venous blood in normal subjects under light pentothal anesthesia.

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